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Transactions of the American Pediatric Society

American Pediatric Society

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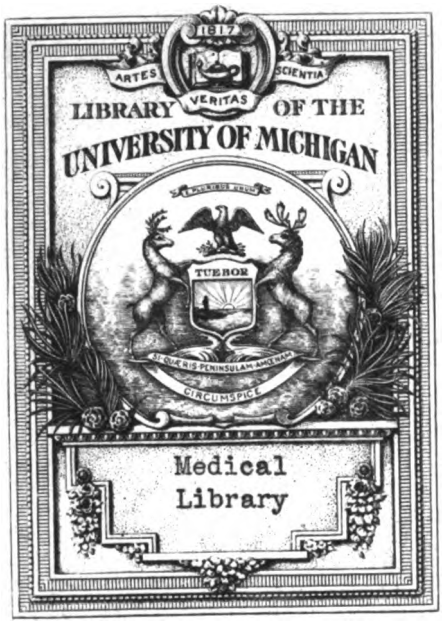

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TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

FIFTEENTH SESSION

HELD AT WASHINGTON, D.C., MAY 12, 13, AND 14, 1903

EDITED BY

WALTER LESTER CARR, M. D.

REPRINTED FROM

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1903

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PRESIDENTS

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. Wm. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.

OFFICERS, 1903

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Secretary..... Samuel S. Adams, M.D.
Treasurer..... J. Park West, M.D.
Recorder and Editor..... Walter Lester Carr, M.D.

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F. Forchheimer, M.D.
J. C. Wilson, M.D.
Floyd M. Crandall, M.D.
T. M. Rotch, M.D.
F. A. Packard, M.D.

MEETING PLACES

1888. Washington, D. C., (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22-25.
1892. Boston, May 2, 3, and 4.
1893. West Point, N. Y., May 24, 25, and 26.
1894. Washington, May 29, and June 1.
1895. Virginia Hot Springs, May 27, 28, and 29.
1896. Montreal, May 25, 26, and 27.
1897. Washington, May 4, 5, and 6.
1898. Cincinnati, June 1, 2, and 3.
1899. Deer Park, June 27, 28, and 29.
1900. Washington, May 1, 2, and 3.
1901. Niagara Falls, May 27, 28, and 29.
1902. Boston, May 26, 27, and 28.
1903. Washington, May 12, 13, and 14.
1904. Detroit, May 30, 31, and June 1.

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F. FORCHHEIMER, M.D.
J. C. WILSON, M.D.
FLOYD M. CRANDALL, M.D.
L. EMMETT HOLT, M.D.
F. S. CHURCHILL, M.D.

MEMBERS

ABT, ISAAC, M.D.....4326 Vincennes Avenue, Chicago
ACKER, GEORGE N., M.D.....913 Sixteenth Street, Washington
ADAMS, SAMUEL S., M.D.....1 Dupont Circle, Washington
BAINES, ALLEN, M.D.....194 Simcoe Street, Toronto
BLACKADER, A. D., M.D.....236 Mountain Street, Montreal
BOOKER, WILLIAM D., M.D.....208 West Monument Street, Baltimore
BUCKINGHAM, E. M., M.D.....346 Marlborough Street, Boston
CAILLÉ, AUGUSTUS, M.D.....753 Madison Avenue, New York
CARR, WALTER LESTER, M.D.....68 West Fifty-first Street, New York
CHAPIN, HENRY DWIGHT, M.D.....51 West Fifty-first Street, New York
CHRISTOPHER, W. S., M.D.....508 Dearborn Avenue, Chicago
CHURCHILL, F. S., M.D.....460 Dearborn Avenue, Chicago
COTTON, A. C., M.D.....1485 Jackson Boulevard, Chicago
CRANDALL, FLOYD M., M.D.....113 West Ninety-fifth Street, New York
DORNING, JOHN, M.D.....111 West Forty-eighth Street, New York
EDSALL, D. L., M.D.....346 South Sixteenth Street, Philadelphia
FORCHHEIMER, F., M.D.....Fourth and Sycamore Streets, Cincinnati
FREEMAN, ROWLAND G., M.D.....205 West Fifty-seventh Street, New York
GRAHAM, E. E., M.D.....1713 Spruce Street, Philadelphia
GRIFFITH, J. P. CROZER, M.D.....1810 Spruce Street, Philadelphia
HAMILL, S. McC., M.D.....1822 Spruce Street, Philadelphia
HAND, ALFRED, JR., M.D.....1724 Pine Street, Philadelphia
HOLT, L. EMMETT, M.D.....14 West Fifty-fifth Street, New York
HUBER, F., M.D.....209 East Seventeenth Street, New York
JACKSON, HENRY, M.D.....309 Marlborough Street, Boston
JACOBI, A., M.D.....19 East Forty-seventh Street, New York
JENNINGS, CHARLES G., M.D.....457 Jefferson Avenue, Detroit
KERLEY, CHARLES G., M.D.....113 West Eighty-third Street, New York

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KOPLIK, HENRY, M.D.....66 East Fifty-eighth Street, New York
LADD, S. MAYNARD, M.D.....220 Marlborough Street, Boston
LA FÉTRA, L. E., M.D.....58 West Fifty-eighth Street, New York
MCCOLLOM, JOHN H., M.D.....745 Massachusetts Avenue, Boston
MARTIN, C. F., M.D.....33 Durocher Street, Montreal
MILLER, D. J. MILTON, M D.....345 South Eighteenth Street, Philadelphia
MORRILL, F. GORDON, M.D.....181 Beacon Street, Boston
MORSE, J. LOVETT, M.D.....70 Bay State Road, Boston
NORTHRUP, WILLIAM P., M.D.....57 East Seventy-ninth Street, New York
OSLER, WILLIAM, M.D.....1 West Franklin Street, Baltimore
PUTNAM, CHARLES P., M.D.....63 Marlborough Street, Boston
RACHFORD, B. K., M.D.....323 Broadway, Cincinnati
ROTCH, T. M., M.D.....197 Commonwealth Avenue, Boston
SAUNDERS, E. W., M.D.....3003 Lafayette Avenue, St. Louis, Mo.
SEIBERT, A., M.D.....114 East Fifty-seventh Street, New York
SHAW, HENRY L. K., M.D.....198 Washington Avenue, Albany
SNOW, IRVING M., M.D.....476 Franklin Street, Buffalo
STARR, LOUIS, M.D.....1818 South Rittenhouse Square, Philadelphia
STENGEL, ALFRED, M.D.....1811 Spruce Street, Philadelphia
TOWNSEND, CHARLES W., M.D.....76 Marlborough Street, Boston
WENTWORTH, A. H., M.D.....294 Marlborough Street, Boston
WEST, J. PARK, M.D.....36 8 Belmont Street, Bellaire, Ohio
WESTCOTT, THOMPSON S., M.D.....1833 Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D.....528 Beacon Street, Boston
WILSON, J. C., M.D.....1437 Walnut Street, Philadelphia
WILSON, W. REYNOLDS, M.D.....1709 Spruce Street, Philadelphia
WINTERS, J. E., M.D.....23 West Thirty-seventh Street, New York
YALE, LEROY M., M.D.....432 Madison Avenue, New York

HONORARY MEMBERS

DR. JOHN THOMSON.....Edinburgh, Scotland.
DR. GEO. F. STILL.....London, England.
DR. HENRY ASHBY.....Manchester, England.
DR. O. HUEBNER.....Berlin, Germany.
DR. ESCHERICH.....Graz, Austria.

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Deceased

JOHN A. JEFFRIES, M.D.,
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.,
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.,
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.,
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.,
Born, October 15, 1827,
Died, January 7, 1898.

JOSEPH O'DWYER, M.D.,
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.,
Born, November 9, 1851,
Died, December 18, 1900.

FREDERICK A. PACKARD, M.D.,
Born, November 17, 1862,
Died, November 1, 1902.

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MINUTES OF THE FIFTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

HELD AT WASHINGTON, D. C., ON MAY 12, 13 AND 14, 1903, IN CONJUNCTION WITH THE AMERICAN CONGRESS OF PHYSICIANS AND SURGEONS.

The following members were present: G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. Baines, M.D., Toronto; A. D. Blackader, M.D., Montreal; W. D. Booker, M.D., Baltimore; W. L. Carr, M.D., New York; H. D. Chapin, M.D., New York; W. S. Christopher, M.D., Chicago: A. C. Cotton, M.D., Chicago; J. Dorning, M.D., New York; D. L. Edsall, M.D., Philadelphia; F. Forchheimer, M.D., Cincinnati; R. G. Freeman, M.D., New York; E. E. Graham, M.D., Philadelphia; J. P. C. Griffith, M.D., Philadelphia; S. McC. Hamill, M.D., Philadelphia; L. E. Holt, M.D., New York; A. Jacobi, M.D., New York; C. G. Jennings, M.D., Detroit; H. Koplik, M.D., New York; D. J. M. Miller, M.D., Philadelphia; J. L. Morse, M.D., Boston; W. P. Northrup, M.D., New York; W. Osler, M.D., Baltimore; C. P. Putnam, M.D., Boston; B. K. Rachford, M.D., Cincinnati; T. M. Rotch, M.D., Boston; E. W. Saunders, M.D., St. Louis; H. L. K. Shaw, M.D., Albany; I. M. Snow, M.D., Buffalo; L. Starr, M.D., Philadelphia; J. P. West, M.D., Bellaire; H. Williams, M.D., Boston; W. R. Wilson, M. D., Philadelphia.

FIRST SESSION, MAY 12TH.

The annual address of the President, entitled A Review of the Work of the American Pediatric Society, was read by Dr. Griffith, of Philadelphia.

Drs. C. F. Martin and F. M. Fry, of Montreal, presented a paper entitled Synopsis on Some of the Renal Lesions in Infancy, which was read by Dr. Samuel S. Adams.

Dr. A. C. Cotton, of Chicago, read a paper entitled Report of a Case of Chronic Nephritis in a Four-Year-Old Boy Having Only One Kidney, and showed photographs.

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Drs. T. M. Rotch and H. H. Cushing, of Boston, presented a paper entitled A Case of Edebohls' Operation, which was read by Dr. Rotch.

Discussion by Drs. Edebohls, Rachford, Jennings, Cotton, Adams and Rotch.

Dr. S. S. Adams, of Washington, exhibited A Case of Club Feet operated on by Professor Lorenz, and showed casts of the case before operation.

Discussion by Drs. Rotch and Adams.

Dr. Adams showed, also, specimens from A Case of Abscess of the Lung.

Discussion by Drs. Rotch, Blackader, Cotton and Adams.

Dr. Simon Flexner, of Philadelphia (by invitation), opened the discussion on The Relation of the Bacillus of Shiga to the Summer Diarrheas.

Discussion was continued by Drs. Park, Koplik, Holt, Knox, Guest, Booker and Flexner.

Dr. Irving M. Snow, of Buffalo, presented a paper entitled Gastroenteric Infections of Newborn Children.

Drs. S. McC. Hamill and W. R. Nicholson presented a paper entitled The Infections of the Newborn, which was read by Dr. Hamill.

Discussion on both papers by Drs. Koplik, Rotch and Shaw.

Dr. R. G. Freeman, of New York, read a paper entitled The Etiology of Rachitis.

Discussion by Drs. Shaw, Adams, Miller and Cotton.

THIRD SESSION, MAY 14TH.

Vice-president Dr. Chapin occupied the chair.

Drs. D. L. Edsall and A. Fife, of Philadelphia, presented a paper entitled Some Chemical Analyses of Milk Mixtures, which was read by Dr. Edsall.

Dr. H. L. K. Shaw, of Albany, read papers entitled (*a*) The Determination of Fat and Total Solids in Milk, and (*b*) A Note on Abdominal Ausculation in Infancy.

Discussion on the paper of Drs. Edsall and Fife and on the first paper of Dr. Shaw by Drs. Rotch, Adams, Cotton, Morse, Hamill, Chapin and Edsall.

Dr. H. D. Chapin, of New York, presented a paper entitled Principles of Infant Feeding as Based on the Evolution of Mammals, with photographs.

Discussion by Drs. Rotch, Koplik and Chapin.

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Dr. J. L. Morse, of Boston, read a paper entitled A Report of Eight Cases of Pneumonia in Infants Treated with Antipneumococcic Serum.

Discussion by Drs. Jennings and Saunders.

Dr. G. N. Acker, of Washington, read a report of A Case of Myxedema, and showed patient.

Discussion by Drs. Kopiik, Rotch, Cotton, Blackader, Adams and Acker.

Drs. T. M. Rotch and H. Dunn, of Boston, presented the history and photographs of A Case of Pulmonary Osteoarthropathy in a Child Three Years Old. The paper was read by Dr. Rotch.

Discussion by Dr. West.

Dr. J. P. West, of Bellaire, showed (*a*) A Case of Chondrodystrophy Fetalis in a Child Fourteen Months Old, with photographs and specimens, and (*b*) A Case of Congenital Hypertrophic Stenosis of the Pyloris, with specimen.

Dr. Koplik, of New York, read a paper entitled The Prophylaxis in the Prevention of the Spread of Vulvovaginitis in the Hospital Service.

Discussion by Drs. Chapin, Cotton, Dodson (Guest) and Dorning.

Dr. W. P. Northrup, of New York, read papers entitled (*a*) Nasal Intubation (soft rubber) for Relief of Dyspnea Due to Acute Nasopharyngeal Swelling in Infants, and (*b*) O'Dwyer Intubation Instruments. Added Tubes for Infants Under One Year.

Discussion on the first paper by Drs. Morse, Koplik, Chapin and Northrup.

Discussion on the second paper by Drs. Dorning, Saunders and Northrup.

Dr. W. R. Wilson, of Philadelphia, read a paper entitled Disturbances of Respiration in the Newborn.

Discussion by Drs. Saunders, Hamill and Wilson.

The following papers were read by title:

Dr. A. Caillé, of New York, A Proposition to Immunize Young School Children Once or Twice During the School Year with Diphtheria Antitoxin and Thereby Lessen the Mortality from Primary Diphtheria, Scarlatina and Measles Complicated with Diphtheria.

Dr. L. E. Holt, of New York, Two Cases of Abscess of the Lung.

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Dr. A. Hand, Jr., of Philadelphia, The Value of the Incubation Period in the Diagnosis of the Contagious Diseases of Childhood.

Dr. F. Huber, of New York, (*a*) Study of Two Cases of Intussusception (Boy of Six and Girl of Nine Years Old), and (*b*) Lumbar Puncture in Serous Meningitis.

Dr. D. L. Edsall, of Philadelphia, Concerning the Nature of Still's Type of Chronic Deforming Polyarthritis.

EXECUTIVE SESSION, MAY 13TH.

The report of the Council was presented by Dr. Blackader.

On nomination of Council the following officers were elected for the ensuing year:

President, - - - - A. CAILLÉ, M.D., New York.
First Vice-President, - A. BAINES, M.D., Toronto.
Second Vice-President, E. E. GRAHAM, M.D., Philadelphia.
Secretary, - - - - S. S. ADAMS, M.D., Washington.
Treasurer, - - - - J. P. WEST, M.D., Bellaire.
Recorder and Editor, - W. L. CARR, M.D., New York.
Members of the Council, { L. E. HOLT, M.D., New York. F. S. CHURCHILL, M.D., Chicago.

Elected to Membership: Maynard Ladd, M.D., Boston; J. H. McCollom, M.D., Boston; L. E. La Fétra, M.D., New York; Isaac Abt, M.D., Chicago.

Elected to Honorary Membership: Dr. John Thomson, Edinburgh; Dr. Geo. F. Still, London; Dr. Henry Ashby, Manchester; Dr. Escherich, Gratz, Austria; Dr. O. Huebner, Berlin, Germany.

Assessment for the ensuing year—*Ten dollars.*

Place of meeting, Detroit, May, 1904.

Upon motion by Dr. Hamill the Society voted to allow the members to publish their papers in any journal, the Society to print its own Transactions.

Upon motion by Dr. Christopher the Society voted to defray the expenses of printing the index to the Transactions.

WALTER LESTER CARR, M.D.,
Recorder.

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A REVIEW OF THE WORK OF THE AMERICAN PEDIATRIC SOCIETY.

PRESIDENTIAL ADDRESS.

BY J. P. CROZER GRIFFITH, M.D.,
Philadelphia.

I wish to thank you, gentlemen, most heartily for the honor which you have conferred upon me in electing me your president; an honor which I feel is as great as it was unexpected.

Realizing that this is the Congress year and that our sessions are necessarily curtailed on this account, while our program is full, it has not seemed to me wisest to take up any medical topic for discussion, or to give you the result of any studies or experience of my own, but to confine myself to the brief expression of a few thoughts which this fifteenth anniversary of our organization as a national society might naturally call to the minds of all of us.

What have we done in the years we have been together? How have we used our talents for the benefit of our race? Probably the most apparent good has been accomplished by our individual care of the thousands of children with whom we have come in contact, and by the hours spent by the bedside in private practice and hospital wards or dispensaries. Often failing and often disappointed, we have yet, over and over, been able to see lives saved, apparently as a result of the treatment we have employed for, and the attention we have given to, our little patients. Physicians may, I think, experience nothing more gratifying and satisfying than the consciousness of work well done and success attained in this way.

Yet, after all, this is not our chief work as a Society. Anything that tends to make the recognition and treatment of disease easier for our brother practitioners, by experimental and clinical studies which we may carry out and by the publication of our results, is capable of doing good far more widely reaching than our individual efforts at the bedside can accomplish. What, then,

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in the fifteen years of our existence have we done, as a body and as individuals, in our attempts to advance the knowledge of pediatrics? An examination of our records, including the papers presented at this meeting, shows a total of 395 articles read before the meetings of the Society by its members and published in the Transactions. To mention the title of any of these as being of particular value would be to make invidious distinctions. Some are reports of cases, some elaborate papers and some descriptions of new instruments. I may say that many of them have been of a nature making them of service to physicians everywhere, and have tended to give our Society a reputation international as well as national, and to make our Transactions of great value to any interested in the subject of diseases of children. Besides these individual efforts we have had some valuable collective investigations conducted by committees of our members, and contributed to by physicians throughout the country. I may mention here the two collective studies of diphtheria and that upon infantile scurvy, all of which have been widely quoted. Committees have also been appointed to simplify, as far as possible, the nomenclature of groups of diseases, such as those of the digestive tract, and the work of these, too, has been of great service. The remarks made by members in the course of discussion have not been included in the figures which I have given, although they have often been of value as great as, if not greater than, that of the articles themselves.

But the literary work of those who are, or who have been, members of the Society has been far more extensive than is represented by the list just given. It has not been possible to determine accurately just how much work of the kind has been done outside of that presented before the Society. As near as I could estimate from a search through medical journals I find that in the fifteen years of our existence, counting from June, 1888, to the present time, physicians who were, at the time of writing, members of our Society have contributed in all 1,535 articles to medical journal literature in addition to those which have been published in the volumes of our Transactions. This by no means includes all the literary medical work that these men have done. I have enumerated only the contributions made while they were members, not the long list of those written before they joined the Society, or, in the case of a number of individuals, the work done after separation from our body. In addition to these journal

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articles those who are, or who have been, members with us have written or edited a number of valuable contributions to text book literature. I can recall at least thirty such works. Some of them have become standard in medicine. Some are almost classic. Notable among the list bearing upon pediatrics is that monument to the name of the editor, "Keating's Cyclopedia of the Diseases of Children." Our members, too, have contributed largely to composite text books, such as the "Twentieth Century Practice of Medicine," the "Reference Handbook of other Medical Sciences," and the like. The two journals in English devoted solely to the subject of pediatrics were established by men at the time members of the Society.

I am sure, gentlemen, that you will not misunderstand what has been said as though it were idle boasting. What the members of the Society have done in the past should only be a stimulus for renewed efforts in the future. Some of our best workers have fallen by the way. Of our members eight have been removed by death, the last of these the lovable Packard, cut off in the midst of his promising labors, of whom those of us who knew him best can say with Hamlet, "He was a man! Take him for all in all I shall not look upon his like again." Still others have finished their labors who had severed their active connection with the Society. I can recall at least three such. Of the present fifty-five members only twenty were in the original list fifteen years ago. Yet probably the majority of us have been members for ten years and more. Ten or fifteen years make a big difference in a man's life. To paraphrase the words of Solomon the Wise, there is a tendency for the sound of the literary grinding to be low and for the grasshopper of scientific investigation to be a burden. Some of us may feel a premature loss of our love of writing, or we may be wrapped up in our busy life in which the needs and opportunities for financial gain may smother our zest for painstaking and laborious scientific medical studies. This must not be. Others besides ourselves are working and have worked. The most scientific labor, take it all in all, is not coming from this country, although the interest in and output of pediatric labor and literature has increased enormously everywhere in recent years. We must keep abreast of the times. As a nation we are by nature almost too practical. More elaborate chemical and bacteriological researches into the causes and treatment of disease in children should receive greater attention from

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us, not neglecting that for which we are well fitted as a nation—the careful study of the clinical side of disease, the exhaustive critical study of the work upon pediatric subjects of others throughout the world, and especially the collective investigations in which the Society as a body has already notably distinguished itself.

Press onward, then, my fellow members, in your efforts for the reputation of our Society, and for the advancement of this branch of medicine, acquaintance with which is so important to physicians, and, through them, to our entire race.

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INFECTIONS OF THE NEWBORN.

BY SAMUEL MCC. HAMILL, M.D.,
Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine,

and

WILLIAM R. NICHOLSON, M.D.,
Obstetrician to the Maternity Hospital, Philadelphia.

We shall limit ourselves in this report to a brief statement concerning infections of the newborn based mainly on our own observations. We do not aim at a definite classification of these conditions; it is rather our desire to condemn certain attempts at classification that have been made. For instance, we do not incline to accept as pathological entities such conditions as melena neonatorum, hemorrhagic disease of the newborn, Buhl's disease, Winckel's disease, hemophilia of the newborn, etc. According to our observations and in accordance with the findings in the literature, all the clinical manifestations described under these various headings may exist as evidences of any one of a number of different infections, the nature and severity of the symptoms depending upon the character and virulence of the infecting organism, and the degree of individual resistance. In the light of our present knowledge, therefore, and for the purposes of simplicity, and to stimulate investigation, we would include all these conditions under the term—infections of the newborn. If any attempt at classification is to be made today, it must be on a purely bacteriological basis.

In order to give a more definite idea of the type of cases which we would include under this heading, we cite a few illustrative instances:—

CASE I.—(Previously reported by one of us.) A male infant, born at term at the Maternity Hospital, of Philadelphia, after a normal labor. He weighed 3,350 grams at birth. The cord separated without incident rather earlier than usual. The infant continued normal until the sixteenth day, when he developed a slight stomatitis. The temperature was normal. On the following day, a slight elevation of temperature and green stools were

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noted. By the next day, there was an almost constant flow of blood from the mucous membrane of the mouth, marked exhaustion, inability to nurse, and bright red blood was passed in the stools. During the succeeding night and the next day (the day of death and the nineteenth since birth), the infant emaciated rapidly and had a series of convulsions which lasted up to the time of death.

The Autopsy was made by Dr. Joseph Sailer. The essential features noted were a hemorrhagic effusion around the umbilicus, a hemorrhage into the wall of the umbilical cord, a hemorrhagic effusion into the peritoneum and intense congestion of the mesentery. The mesenteric lymph nodes were not enlarged and there were no ecchymoses. The spleen was normal. The ileum contained altered blood. Neither the intestinal tract nor the stomach showed congestion or ulceration. The esophagus was filled with blood-stained mucus. The liver was enlarged, soft, and its appearance on section was distinctly cloudy. The adrenals and kidneys were normal. The brain was edematous and its membranes very cloudy, but otherwise normal.

An extensive histological examination was made by Dr.Sailer, which may be summed up as follows:—There was a generalized parenchymatous degeneration of the organs, a sclerosis of the pancreas, an acute enteritis and a commencing cirrhosis of the liver. Cultures were made at the autopsy from the heart's blood, left lung, spleen, liver, peritoneum, brain, contents of the ileum and the bile. The staphylococcus pyogenes aureus was recovered from the heart's blood, spleen and brain; the bacillus aerogenes lactis was determined in the heart's blood, spleen, peritoneum and in plates made from the contents of the ileum. From the bile were recovered the bacillus aerogenes lactis and a typical growth of the bacillus pyocyaneus. From the liver tissue, the three microorganisms mentioned were obtained. All three microorganisms, when isolated, proved pathogenic to guinea-pigs.

CASE II.—(Previously reported by one of us.) For the clinical notes of this case we are indebted to Dr. Bolles, resident physician of the Maternity Hospital. The infant was born after a normal labor on the 10th of January, 1902. The child was not asphyxiated; there was no history or evidence of syphilis in the mother. His temperature was slightly elevated from the second day; on the seventh day he developed a papular eruption on his face. His temperature rose suddenly with the appearance of the eruption to 104° F. He took nourishment well and had no

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gastrointestinal disturbance. His temperature fluctuated between 101° and 104° F., from the eighth to the twelfth day. The facial eruption persisted, and a similar eruption developed over the neck, shoulders and flexor surface of the arms. The persistent fever was attributed to this eruption.

On the thirteenth day "the baby developed symptoms suggestive of pneumonia"—rapid respiration and a harsh vesicular murmur. The temperature became a little more elevated. A cotton jacket was applied.

On the fourteenth day, marked twitching of the muscles of the right eye and constant lateral rotation of the head were noted. There were no general convulsions. The child did not cry out. He nursed poorly and was somewhat cyanosed.

On the fifteenth day the temperature was less high, 102° F., and he seemed generally better.

On the sixteenth day, the day of death, the temperature rose to 106.8° F. at 8 A.M. The child vomited a large quantity of blackish material, which was evidently altered blood, and had several tarry stools. The face became much cyanosed; great restlessness ensued, and death occurred rather suddenly at midday.

The Autopsy was made by one of us twenty hours after death. A papulopustular eruption was present on the face, neck, forehead, shoulders, arms and upper part of the chest, in some areas it was hemorrhagic, especially over the flexor surfaces of the arms. The umbilical stump was not entirely healed, and rather unhealthy in appearance.

Thorax.—Both auricles were distended with liquid blood, the right more than the left. The veins of the heart were greatly distended. The lungs showed some congestion at their margins.

Abdomen.—The umbilical arteries contained thrombi. The principal lesions were some congestion of the vessels of the mesentery and enlargement and congestion of its lymph nodes.

The stomach contained some blackish material having the appearance of altered blood. There was no congestion nor erosion of its mucous membrane. The intestines contained nothing abnormal. The liver on section was yellowish in color and rather soft. The spleen was moderately congested. The kidneys were anomalous.

Brain.—On opening the cranium, following the lines of the sutures, there was found, overlying the left hemisphere, a large mass of clotted blood, more marked, anteriorly. This extended

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into the substance of the brain causing some destruction of tissue. Overlying the right hemisphere there was a less marked hemorrhage. The superior longitudinal, the left lateral and the straight sinus and the vena magna Galeni contained firmly organized thrombi. The veins of the brain throughout were distended with blood, and some of the superior cerebral veins on the left side contained thrombi; both lateral ventricles were dilated and contained considerable blood, especially the left.

Cultures were made on blood serum from the thrombus in the umbilical artery and from the spleen. There was a moderate growth on each tube. Cultures from the heart's blood were contaminated. Agar slant transfers showed profuse growth after twenty-four hours; one showed a pure growth and the other an almost pure growth of a bacillus, possessing practically the same cultural characteristics as the colon bacillus, although not absolutely typical.

CASE III.—The clinical notes of this case were also prepared by Dr. Bolles. The infant was born on April 3d, in the Maternity Hospital, after a labor with a prolonged first stage. It showed no abnormality, but was poorly nourished and small. It nursed well at first. On the fifth day there was some oozing of blood about the matrix of the nail of the index finger of the right hand. On the same day it became jaundiced. On the sixth day it had a hemorrhage from the mouth while nursing and profuse bleeding from the first finger and thumb of the right hand and later in the day from all the fingers, together with some bleeding from the umbilicus. The infant cried and moaned constantly, apparently from pain. The jaundice became more marked and the breast was refused. During the following night there was a severe hemorrhage from the vagina, the rectum and the umbilicus. During the seventh day a subcutaneous hemorrhage appeared over the middle third of the right tibia. Two convulsions occurred during the following night. The hemorrhages continued the following day and it was noted that the napkins were "soaked through." Emaciation became rapid and the jaundice more intense. There were some hemorrhages from the corneal vessels. The infant had several convulsions during the day; it became rapidly weaker and died after an extraordinarily profuse hemorrhage from the navel at 11 P.M.

Autopsy was made by one of us eleven hours after death. The principal lesions were subcutaneous hemorrhages over the

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extensor surface of the left.thumb, the anterior surface of the left wrist and the outer surface of the tibia.

Abdomen.—The mesenteric lymph nodes were enlarged and congested and the mesenteric vessels were full.

The intestines and stomach contained, throughout their course, a tarry substance resembling altered blood. There was moderate inflammation of the mucosa. There were several small hemorrhagic areas in the large intestine but no ulceration and no enlargement of the follicles. The bladder contained some bloody fluid. The vaginal mucous membrane was pale and there was no evidence of hemorrhage into the uterus.

The liver was enlarged and yellowish on its surface and on section. The left kidney showed an extensive hemorrhagic area limited to the anterior surface. There was no hemorrhage into the pelvis of the left kidney. The hemorrhagic area referred to was limited to the cortex. There was slight congestion of the left suprarenal. The right kidney showed numerous hemorrhagic areas throughout the medulla and cortex. There was no hemorrhage into the pelvis. The right suprarenal was more congested than the left and there was a slight hemorrhage into the medullary portion. The spleen was moderately enlarged and much congested. There were no thrombi in the renal vessels.

Thorax.—There were numerous small hemorrhagic infarcts scattered through both lungs. There was some clotted blood in the larger branches of the right bronchus. The thymus gland was larger than normal but not congested. There was a small amount of fluid in the pericardium which was unusually yellowish in color. There was nothing abnormal about the heart.

Brain.—There was considerable congestion of the veins of the brain, especially over the right hemisphere. There was very little blood and no thrombi in the sinuses. There was no hemorrhage into the ventricles nor into the substance of the brain. Whether or not the bloody urine was a hematuria or hemoglobinuria was not determined. The case bore a close resemblance to socalled Winckel's disease.

Cultures made from the heart's blood, the clot in the umbilical artery and from the spleen showed a pure growth of the staphylococcus aureus. The microscopic examination of the liver, kindly made by Dr. C. Y. White, showed some fatty infiltration, together with some fatty degeneration of the cells. Microscopic examination of the long bones showed no evidence of syphilis.

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CASE IV.—Baby Young. Born on April 7, 1903, in the Maternity Hospital, after a normal labor, lasting eleven hours. The previous and present health of the mother was good. The father was healthy. There was no history of syphilis.

The temperature was elevated to 102° F., at birth. The condition of the infant was not brought to our attention by the hospital resident until about twenty-four hours before death. The stools had been abnormal since birth. On the third day, it was noted that they were greenish in color and slimy. The child nursed poorly. On the fifth day a small cyst, containing clear fluid, was observed in the sublingual region. The cyst had increased in size by the following day and the character of the stools remained unchanged. On the morning of the seventh day (the day of death), the child developed nystagmus and marked twitching of the muscles of the mouth. There was a slight bloody discharge from the lower bowel. The diarrhea became more marked. There were no hemorrhages from any other part of the body. The child became increasingly cyanotic. The pupils contracted; the cornea became somewhat opaque. Small papules and pustules were noted on the neck and face. The child moaned constantly and refused to nurse. Respirations became increasingly difficult; almost persistent marked contractions of the muscles of the neck and lower jaw developed with intermittent general convulsions. Death occurred in the middle of the afternoon.

The Autopsy was made by one of us eighteen hours after death. The chief points of interest were a papular and papulopustular eruption on the forehead, cheeks, neck and shoulders. There were ecchymotic areas on the neck. No eruption elsewhere. The umbilical stump had an entirely healthy appearance.

Abdomen.—The mesentery was congested. The lymph nodes were enlarged and deeply cyanosed. The stomach contained a moderate amount of altered blood. There was decided congestion of the mucosa in the pyloric and cardiac areas; a few hemorrhagic areas but no ulceration. There was moderate congestion in the lower part of the ileum and one or two Peyer's patches were congested. The most marked congestion, however, was in the colon. This extended throughout its entire length, being especially evident in the upper part of the sigmoid and in the descending colon. There was marked enlargement of the isolated follicles and many ecchymotic areas, but no naked eye evidences of ulceration. The spleen was enlarged and purplish on section. The liver was en-

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larged and mottled, yellowish purple on the surface and on section. Both kidneys were congested, especially the right, the latter containing some hemorrhagic infarcts. The right suprarenal gland was congested and the left hemorrhagic. The bladder was empty and there was no congestion or hemorrhage into its mucous membrane.

Thorax.—The bronchial and all the mediastinal lymph nodes were enlarged and deeply congested; some of them almost hemorrhagic. Both lungs were generally congested but crepitant throughout. Both auricles and the vena cava were distended with liquid blood.

Cranium.—The amount of cerebral fluid was increased above the normal. There was considerable fullness of the vessels at the base of the brain and moderate fullness of the superior cerebral veins. The sinuses were empty.

Cultures were made from the heart's blood, spleen, umbilical cord and lungs. A pure culture of the staphylococcus pyogenes aureus was obtained from the heart's blood, umbilical cord and the lungs. Cultures from the spleen were negative. Guinea-pigs were inoculated from a first subculture. The organism did not prove pathogenic, probably owing to the well-known rapid deterioration in the virulence of this particular microorganism.

CASE V.—Baby Letters. Born at Preston Retreat, after a normal labor on the 17th of February, 1903. The temperature was normal at birth. It rose to 101° F. the following day and continued above normal until death. On the third day, the temperature became suddenly elevated to 105° F. The child developed rapid (60 per minute) labored respirations and cyanosis. The cyanosis varied in its degree of intensity from time to time. The pulse rate increased with the respirations. On the fifth day, the child had green, mucous, undigested stools. Up to this time, there had been no intestinal symptoms. There were no hemorrhages at any time and no convulsions or muscular twitchings. The cyanosis and rapid respirations increased persistently up to the time of death on the seventh day and led to the diagnosis of pneumonia.

The Autopsy was made by one of us ten hours after death. There were no skin eruptions. The umbilicus was healthy in appearance.

Abdomen.—The umbilical vessels contained clots. The mesenteric vessels were full. There was marked congestion of the

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omentum. The large intestine was quite empty; the upper part of the small intestine and the stomach contained a bluish substance. On opening the colon, the mucous membrane was found extremely congested and in some places hemorrhagic. The same conditions existed in the small intestine. The blackish substance in the upper part of the small intestine and in the stomach was altered blood. The mucous membrane of the stomach was less congested than that of the intestines. The spleen was normal, the liver congested.

Thorax.—The left pleural cavity contained about a tablespoonful of clotted and liquid blood. There were no adhesions between the parietal and visceral pleuræ. The left lung contained numerous large hemorrhagic infarcts. The right lung was almost completely atelectatic. There was one hemorrhage infarct at its base, posteriorly. The bronchial lymph nodes were hemorrhagic. The minute vessels of the visceral and parietal pleuræ were very full. The point of origin of the hemorrhage could not be determined. The thymus, heart and vessels were normal. Examination of the brain was not permitted.

Cultures were made on agar-agar slants and in litmus milk from the clots in the umbilical vessels, the liver, the spleen, the blood in the pleural cavity and the heart's blood. Bouillon cultures were made also from the heart's blood. All these cultures were negative except the bouillon cultures from the heart blood. From this was obtained a pure growth of the bacillus coli immobilis. This microorganism, injected subcutaneously and intraperitonially, proved pathogenic to guinea-pigs, causing diffuse subcutaneous hemorrhagic infiltration, hemorrhagic peritonitis, pleuritis and pericarditis, and enlargement and congestion of the liver, spleen and suprarenals. The same microorganism was obtained in pure culture from all the organs and the serous cavities.

There is one other case in our series, which showed much the same lesions and in which the bacillus coli immobilis was isolated. It was also pathogenic to guinea-pigs causing similar lesions.

CASE VI.—Baby Smart. Was born in the Maternity Hospital on the morning of April 17, 1903. The temperature was normal at birth and had fallen to 97° F., by the evening of the 17th. It became elevated to 101° F., on the morning of the second day and continued above normal during the remainder of life, fluctuating between 99° and 104° F. On the third day the stools became yellowish-green and loose, and contained mucus. The

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child lost weight persistently from birth, but aside from the persistent temperature and correspondingly increased pulse and respiration rate and the character of the stools, nothing else abnormal was noted until the evening of the 26th of April (ninth day), when dusky red spots developed on the lower anterior portion of the tibia and on the extensor surface of the forearms. These areas rapidly became indurated and extended with marked rapidity involving, by the following morning, both cheeks, the buttocks, the flexor surfaces of the feet, the extensor surfaces of the left arm, the fingers of the left hand, but not the thumb. The areas on the face were circular in character, extending from above downward, from the lower lid of each eye to the base of the lower jaw, and antero-posteriorly from the nose to the ear. All of these areas became indurated. The redness disappeared on pressure of the finger, to return again quickly, there was no pitting and the margins were distinctly limited. During the course of the day, the index finger of the left hand became bluish black, and, by the following morning (the 28th), all of the fingers, a portion of the extensor surface of the left hand and, in fact, all of the indurated areas, were gangrenous. There was no change in the character of the fever at this time. The child emaciated rapidly and, on the evening of the 28th, had a slight intestinal hemorrhage. The respirations became labored, muscular twitchings developed on the 29th and after midday the infant developed convulsions, which persisted until death, on the morning of April 30th (the thirteenth day).

The areas of induration described, aside from their color, closely simulated sclerema neonatorum. The area of distribution was suggestive, but the failure to spread after a period of ten or twelve hours was not entirely characteristic. Blood cultures were made during life, prior to the development of the condition just described, from a vein in the left forearm. Later cultures were made from blood obtained from an incision made over one of the areas of induration. At the time of this incision, it was noted that the surface redness was very superficial; that the deeper tissues were distinctly whitish in character and had the appearance of hardened subcutaneous fat.

Autopsy.—A postmortem examination was obtained, only after much arguing with the father, and was limited absolutely to an abdominal incision. This incision was, however, extended sufficiently to enable us to expose the heart and to obtain cultures

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from the heart's blood. The principal changes noted were a mottled yellowish purple color of the liver, a normal spleen, moderate enlargement and congestion of the mesenteric lymph nodes, and small clots in the umbilical vessels; there was moderate congestion of the mucosa of the lower portion of the large intestine. There was some altered blood in the stomach but no inflammation of its mucosa. The small intestine was normal. The bronchial and the mediastinal lymph nodes were deeply congested. Cultures were made from the spleen, the lungs, the umbilical vessels and the heart's blood. In all of the cultures made during life and in the cultures made from the heart's blood, postmortem, there was found the staphylococcus aureus and a streptococcus.

ETIOLOGY.—Hemorrhagic conditions in the newborn may, in certain instances, be due to some of the causes formerly held accountable for all such manifestations. Among these may be mentioned birth trauma, fetal malformations, asphyxia and syphilis. In the vast majority of instances, however, hemorrhages may be considered as symptoms of an infectious condition.

Strong evidence in favor of the infectious nature of all such conditions, as melena, Winckel's disease, etc., rests in the fact that they are observed almost exclusively in institutions and not uncommonly in epidemic form. One peculiar feature of this last observation is that bacteriological studies, while proving the cases infectious, have commonly failed to demonstrate the presence of the same microorganisms in all of the cases, a result which suggests that these infections are more often dependent upon the deficient technique of a poorly trained nurse than upon defective conditions in the ward.

The nature of the infecting organism varies widely. In the 6 cases we record, six different microorganisms were isolated, viz.: the bacillus pyocyaneus, the bacillus lactis aerogenes, the colon bacillus, the staphylococcus aureus, the bacillus coli immobilis, and a streptococcus. In our complete list of cases, amounting to about 15, only one other microorganism, an unclassified micrococcus, has been encountered. The literature, however, contains instances of infection by the pneumococcus, Pfeiffer's bacillus, the bacillus of Babés, the bacillus hemorrhagica of Kolb, the bacillus of Gaertner, the encapsulated bacillus of Dungern, the pneumococcus and a bacillus closely resembling the pneumococcus, but differing from it in certain essential characteristics, (Kilham

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and Mercelis). Of these various microorganisms, the streptococcus, the bacillus coli commune and the staphylococcus are the ones which have been most commonly encountered. Delestre, for instance, in a series of ante and postmortem blood cultures made from infected infants, premature and born at term, obtained positive results in thirty-seven. In this number, the streptococcus was found fourteen times, the colon bacillus ten times, and the staphylococcus six times.

THE ORIGIN OF THESE INFECTIONS.—As has been said, these infections occur most frequently in maternity hospitals. The opportunities for the dissemination of disease germs in institutions are extraordinarily great. Without the utmost precaution, their presence is unavoidable. Bacteriological investigations have shown the presence of various microorganisms in the air and dust of the wards, especially in the region of the clothing from the patients and their beds. The air is probably infected through the medium of the clothing and hands of attendants, the stools and buccal secretions of the patients, the bed clothing and the infants' napkins. It is possible, therefore, that air infections do sometimes occur.

The mother's milk has been held accountable and doubtless is responsible for a small percentage of cases. In the presence of suppurative lesions of the breast pathogenic microorganisms are most liable to be introduced. In milk from apparently normal breasts microorganisms, mainly the staphylococcus aureus and albus and the streptococcus, have been isolated. The nature of these microorganisms and the fact that in the udder of the cow concealed foci of suppuration are not uncommon, would suggest the likelihood of a similar condition in the human breast.

A source of infection that has been noted is the bath water of the infant. In certain instances, recorded in the literature, the use of the same water for the ablutions of several infants has resulted in infecting the water; bacteriological studies of such water have shown the presence of some of the microorganisms mentioned. The placing of infants in beds which have been previously occupied by infected infants and the bedding together of the infected and noninfected, has resulted in conveying the infection. Admitting, as we do, the accountability of all these sources, we are inclined to believe that the most common medium of infection is the poorly trained or careless nurse. In the institutions in which we have observed these cases, even when they have occurred

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in epidemic form, we have noted that the cases have not been confined to any one ward; that they have been handled by the same nurse and that the bacteriological studies have shown the presence of different microorganisms in the different cases. The records in the literature show very few instances of epidemics in which any one microorganism has been held accountable for all the cases. These facts, we think, argue against the greater frequency of air infections.

It is impossible to definitely fix upon the port of entry. The statements just made, together with our general observations, lead us to believe that the buccal cavity, the tonsils, pharynx and the remainder of the alimentary tract, is the most common. Next in order we would implicate the lungs. We believe that the cord has been given too great prominence as the point of entrance and that the other avenues named—the skin, conjunctiva, nose, ears and urogenital tract—are rarely responsible.

POSTMORTEM APPEARANCES.—The appearances observed postmortem may be summed up in the words "congestion" and "hemorrhage." These vary in great degree in different cases. In some instances there are no hemorrhages whatsoever, but one rarely fails to find a considerable degree of congestion of the spleen, the mesenteric lymph nodes, the kidneys, suprarenals, liver, stomach or intestines. There is usually marked fullness of the mesenteric vessels; sometimes amounting to hemorrhage. The mucosa of the stomach and intestines, especially that of the large intestine, in many cases, is inflamed and thickened. The isolated follicles and Peyer's patches may or may not be enlarged. Ulceration in various portions of the gastrointestinal tract has been recorded. Ulceration of the duodenum just below the pylorus was observed by Landau and by him attributed to the blocking up of the gastroduodenal artery by an embolus, originating from a thrombus in the umbilical vein. Two similar cases have been reported by Kling. The liver, the kidneys and the suprarenal glands may be the seat of small or large hemorrhages. Instances have been recorded in which a hemorrhage into these organs or into the perirenal tissues has secondarily ruptured into the peritoneum, giving rise to profuse fatal hemorrhage. Bloody effusions into the serous sacks are of frequent occurrence. The bladder sometimes contains bloody urine, its mucous membrane may be inflamed and sometimes ecchymotic.

The changes commonly noted in the thoracic organs are:

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marked enlargement and congestion of the mediastinal nodes, various degrees of congestion in the lungs, hemorrhagic infarcts, limited areas of, or sometimes, complete atelectasis and occasionally pneumonia. The thymus is frequently congested, and may be hemorrhagic. The right auricle and ventricle ordinarily contain liquid or partially clotted blood. The heart muscle is sometimes soft and its vessels are frequently enormously distended. The blood in some infections seems to retain its fluidity to a remarkable degree, although instances of antemortem clotting have been noted. Thrombosis and embolism are common.

Brain.—The changes consist of various degrees of thickening of the membranes, moderate or severe congestion, subdural hemorrhage, sometimes hemorrhage into the substance of the brain or into the ventricles. Extensive thrombosis of the sinuses and acute hydrocephalus have been noted.

Histological studies have been comparatively few and incomplete. The principal changes are those commonly found in infectious conditions; namely, cloudy swelling, fatty degeneration and infiltration of the liver and kidneys, in addition to the various changes resulting from hemorrhage.

SYMPTOMS.—These infections may occur during, before or after labor. Most often they occur within the first week of life, although their onset may be delayed beyond the fifteenth day. Their degree of severity seems to differ according to the virulence of the infection. Profound toxic symptoms, suggesting disease of special organs, such as the lungs, may occur, the autopsy revealing absolutely no pulmonary lesion. On the other hand, the course of the infection may be attended by few symptoms. The symptoms have no definite order of occurrence. Fever is usually early manifested, and may vary from a slight elevation to hyperpyrexia, 108°F. having been recorded in one of our cases. Diarrhea, with malodorous, undigested, greenish, mucous stools, is present in the majority of cases. Icterus may occur and is sometimes intense. Skin eruptions of almost every description have been noted. The most common are: petechia, ecchymoses, bullæ, erythemata, erysipelatoid eruptions, and especially a papular or papulovesicular eruption, frequently going on to pustulation and involving the face, neck, shoulders and flexor surfaces of the arms. Apathy, rapid and persistent emaciation and inability or disinclination to nurse are always noted. Hemorrhage of varying degree, from either the skin, umbilical cord, eyes, ears, nose,

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mouth, vagina, bladder, stomach and bowels usually happens at some time during the course of the disease. Nervous phenomena are varied; they consist mainly of convulsions, retraction of the head, nystagmus, twitching of the muscles, tetanic and tonic spasms, strabismus, changes in the pupils, and sometimes paralysis. Cyanosis and rapid, labored and irregular respirations, usually toxic in origin, may occur early or, more frequently, late in the disease.

DIAGNOSIS.—But little need be said in relation to the diagnosis of these conditions. Any decided elevation of temperature, notwithstanding the frequency of fever at this age, should arouse suspicion. It may be the only evidence of the condition for several days. In most of the cases we have seen, enteritis has been an early symptom, and next in order, perhaps, is the occurrence of the papular or papulovesicular eruption involving the skin of the face, neck, shoulders and forearms. If, therefore, one observes this combination—fever, greenish, mucous stools, a skin eruption of the character described, and rapid emaciation—one is justified, from the therapeutic standpoint, in considering the condition infectious. If to these manifestations are added hemorrhage, nervous phenomena, cyanosis, and rapid, irregular or labored respiration, the evidences of an infectious condition are complete. Confirmatory data and definite information as to the character of the infection can be obtained by cultures made from the blood.

Several of the cases which we have had the privilege of studying only after death had been diagnosed inspiration pneumonia, this conclusion being based on the presence of fever, labored and rapid respiration, harsh respiratory murmur and cyanosis. Given the ordinarily accepted diagnostic symptoms of pneumonia in the newborn, rapid, labored respirations, recession of the chest wall and cyanosis, because of the comparative infrequency of pneumonia, the assumption that the condition is a blood infection rather than a true lung involvement, is justifiable. The differentiation between these conditions on the basis of the symptoms and physical signs, is not possible.

THE PROGNOSIS of these infections is distinctly bad. Mild infections do occur and, in some instances, the patients recover when the condition seems hopeless. It is a not uncommon occurrence, in private practice, to see mild cases of socalled melena recover, but in institutions the mortality from infections of any type is extraordinarily high.

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PREVENTION.—In the light of what has been said in reference to the source and mode of entrance of these infections, it is evident that their prevention is, in large part, possible, and that this is to be accomplished by measures directed toward the improvement of the general aseptic technique of our maternity hospitals. We would recommend that, in the construction of maternity hospitals, every effort be made to insure ease of cleanliness. Only the necessary attendants should be admitted to the wards. They should wear overgowns or large aprons and cuffs, which should be changed daily. Each morning before entering the wards their hands should be scrubbed for at least five minutes with soap and water, and washed again immediately after the changing of beds, the changing of napkins, or handling of the bed pan, and always before handling the infants. The general cleanliness of the nurse, especially the proper toilet of the mouth, should be insisted upon. The infants should be kept in separate wards from the mothers and be in the charge of a special nurse. The resident and attending physicians should follow the same precautions suggested for the nurse.

We would recommend that the cord be tied, as is usual, before it is severed, with an aseptic ligature by aseptic hands, and that it be immediately dressed with a sterile dressing. We question, to some extent, the advisability of the routine measure of cleansing the infant's mouth, unless it is done under the most aseptic conditions.

The nurse who has handled the bedding of a patient, treated a fissured breast, emptied a bed pan or changed a napkin, unless she be most careful in preparing her hands, will readily infect the mouth of the infant. The condition of the breasts of the mother should be carefully studied. The recognition of erosions, fissures, or any other pathologic condition, should result in the immediate withdrawal of the infant. The common custom of suckling the infant under these conditions is to be strongly condemned. The normal breasts and surrounding areas should be most carefully cleansed before and after each nursing. Infants should not be permitted to suckle mothers suffering from puerperal septicemia. When cases of infection in the mother or infant occur, the wards which they have occupied should be closed and fumigated.

MEDICAL TREATMENT.—Infection once established, there is comparatively little that can be done for these patients in the way of treatment. In an extensive epidemic of streptococcic infec-

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tion, occurring in the Maternity Department of the University of Pennsylvania Hospital a few years ago, it was accidently discovered that two of the infants suffering from infection, when sent to their homes, rapidly recovered. It has been recorded, as a measure of treatment, that isolation of these cases, in hospitals, has a beneficial effect upon the patients. It is desirable, therefore, to isolate these infants as soon as suspicious symptoms arise, placing them in bright, sunny, well-ventilated rooms. Delestre, in making a series of blood cultures from infections of the newborn, under the direction of Hutinel, noted, to his great surprise, that the withdrawal of 2 or 3 c.cm. of blood resulted in some improvement of the condition of the infant. Encouraged by this fact, he withdrew from some infants from 15 to 20 c.cm. of blood, immediately injecting, subcutaneously, 20 to 30 c.cm. of artificial serum, with very beneficial results. He makes the following statement: "I have seen infants cyanosed, cold and practically moribund, resuscitated, positively, under my eyes as a result of this measure." He accounts for these results by the fact that the withdrawal of the blood acts in stimulating the activity of the hematopoietic and lymphpoietic organs.

We have had no experience with this measure. Judging from the difficulty one encounters in securing sufficient blood to make satisfactory blood cultures, one would naturally question the ability to secure this quantity of blood at a single bleeding, as Delestre claims to have done. In the light of the great mortality of these cases, and of the extravagant claims he makes for it, one would be justified in applying it.

The medicinal treatment is the same as that applied to all cases in which we are unable to influence the etiological factor, namely, the treatment of the symptoms. All infectious conditions, especially in those who are possessed of such low resistance, require vigorous stimulation and good nourishment. In some instances we have used high intestinal injections without beneficial results.

NOTE.—The bacteriological examinations in cases 4, 5 and 6 were kindly made for us by Dr. Joseph S. Evans, Associate in Bacteriology in the Pepper Laboratory of Clinical Medicine.

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INJURIES AND INFECTIONS OF NEWBORN CHILDREN.

BY IRVING M. SNOW, M.D.,
Buffalo, N. Y.

In the consideration of this subject we must first study

INFANT MORTALITY IN THE FIRST MONTH OF LIFE.

The first four weeks may be regarded as the most dangerous period of human life; nearly one-tenth of the race succumb in the first month of existence. As a result of scientific midwifery and strict asepsis, puerperal fever has practically disappeared; yet, as I shall be able to prove to you, the morbidity and mortality among young infants remains lamentably high. Beside the injuries of parturition and asphyxia the infant is far more susceptible to infections than the mother. Sudden death is very common, often due to unrecognized intracranial hemorrhage, atelectasis, or pneumonia.

Eröss massed the vital statistics of sixteen large capitals of Europe, including 1,439,000 births. He found that 130,610, 9.5 of all children born, died in the first four weeks of life. In Budapesth, where the mortality in the first month is 8.11, he discovered that of these children 37 per cent. died in the first week, 29 per cent. in the second week, 21 per cent. in the third week, and 13 per cent. in the fourth week of life. The deaths in the earlier days were principally due to malformations, injuries of parturition, and asphyxia. After the first week infections caused the majority of fatalities.

Axel-Johannessen states that the susceptibility to disease lessens with each month of life; the victims of congenital weakness, malformations, injuries of parturition die first and the survivors continually gain resistance to disease.

The following are Norwegian statistics. (Mortality by months of the first year of life.)

1st month	— 3.41 per cent.	4th month	— .67 per cent.
2d "	— .99 " "	5th "	— .62 " "
3d "	— .76 " "	6th "	— .56 " "

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7th month	— .52 per cent.	10th month	— .45 per cent.
8th "	— .50 " "	11th "	— .41 " "
9th "	— .47 " "	12th "	— .40 " "

9.80 per cent. in the first year.

The same excessive death rate exists in Buffalo. During the year ending December 1, 1902, there was 7,290 births and 5,080 deaths. Of the deaths 471 were in children under one month. Thus, 9.30 of all children born in Buffalo die in the first four weeks, and the death rate in the first months forms 6.4 per cent. of the total mortality. Of these children

182	—38.5 per cent.	—died	in the first 24 hours	
141	—30.0 "	" —	" 2d-7th day of life	
78	—16.5 "	" —	" 2d week	" "
34	— 7.3 "	" —	" 3d "	" "
36	— 7.6 "	" —	" 4th "	" "

It may be said that this is a normal mortality, that a large number of children are born who are incapable of living, but this position is erroneous. If we turn to the statistics of Norway (which has the lowest infant mortality in the world) we find that among 1,000,000 births in the rural districts from 1876-1898 the death rate in the first month is 3.41 per cent., about one-third of that of Buffalo. The records of a Norwegian country parish, Gloppen, have been carefully kept for 200 years. From 1687-1711 the mortality in the first month was 12.87 per cent.; it has now fallen to 3.38 per cent., a diminution of about three-quarters. In the city of Prague the mortality in the first month is 14.2 per cent.; in the Prague Hospital it is 3.3, showing conclusively that the life of the newly born child is most favorably influenced by good midwifery and nursing.

In contrasting the statistics of Buffalo with those of Eröss we see that the mortality in the first month is 9.3 per cent., which is about the same as in the cities of continental Europe, 9.5 per cent.; but in comparing the length of life, we notice that nearly double the number, 68.5 per cent., die in Buffalo in the first week, to 37 per cent. in Europe. The number dying from the seventh to twenty-eighth day in Buffalo is 32.5 per cent., in Europe 63 per cent. We may, therefore, draw the conclusion that more children die with us from accidents of parturition and fewer from infections than in Europe.

As to the causes of death the malady or condition is often so

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vaguely described, that it is evident that a confusion of ideas exists. In Eröss' report, congenital debility is the most common term, claiming 80 per cent. of deaths in the first month. It is, however, noteworthy that in Cologne in 1866-68, 5.20 per cent. of all children born were recorded as dying of congenital debility, and in the next decade only 0.7 per cent. were returned under the same title, indicating that the Cologne physicians had changed their phraseology.

Without doubt many infants are born before the full period of gestation, but they die of atelectasis, infections, meningeal hemorrhages. Absence of lesions does not indicate absence of microbes, which may be found in the fluids of apparently healthy tissues. A very large number of prematurely born, underweight babies, reported as dying of immaturity, die of pulmonary or gastroenteric infections, or of cranial hemorrhage.

The mortality in early life is due to: (1) immaturity; (2) malformations incompatible with life; (3) asphyxia and atelectasis; (4) injuries of parturition and (5) various infections.

There were reported in Buffalo for the year ending December 1, 1902, sixteen deaths from malformations, principally of the head and spine; nineteen deaths from congenital heart disease. This hardly seems creditable as patent foramen ovale and ductus arteriosus are compatible with prolonged existence. Cyanosis as a symptom of congenital heart disease is also seen in imperfect expansion of the lung and various infections.

Of children under 1 month, there died in Buffalo in 1902:—

From injuries of parturition,	21 deaths	— 4.5	per cent
" convulsions - -	25 "	— 5.3	" "
		9.8	" "

The conditions of asphyxia and intracranial hemorrhage are most closely associated. The effect of prolonged difficult labor and abnormal presentations, not only favor the aspiration of foreign substances into the lungs and paralyze the medullary respiratory centres with CO_2, but they also cause cerebral hemorrhages from traumatism, as in the application of the forceps, by the nipping of a cerebral sinus in the forcible moulding of the head. Further, the intense cerebral congestion of dystocia may cause a rupture of the very fragile vessels of the pia mater. Hemorrhage is, in

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fact, the most frequent lesion found in infants still born, or those that die soon after birth.

Dr. Herbert Spencer made a critical examination of 130 infants dying in the first few hours of life. In 65 per cent. he found injuries to the brain, congestion and hemorrhages. He considers the forceps as the most frequent agent in producing hemorrhage. next, foot and breech presentations, softness of the skull and relaxation of sutures. He also discovered in many cases hemorrhages into the liver and suprarenal capsules, and pulmonary apoplexies. It is probable that a large number of children returned as dying of immaturity, insufficient vitality, asphyxia and convulsions succumb to intracranial hemorrhage.

It must be remembered that while cerebral hemorrhage is usually associated with dystocia, it may occur in small children who are easily born. There may be no sign of cerebral irritation. The infant may die suddenly without symptoms, or may show irregular respiration and slight cyanosis, simulating asphyxia or atelectasis. Mistaking the condition for an imperfect expansion of the lungs, the physician may wonder why his well-directed efforts at resuscitation are unsuccessful. Generally, meningeal hemorrhage is the result of malpresentations and difficult labor. There is a series of convulsions, irregular breathing, cyanosis. opisthotonus, rigidity, quivering and automatic movements. The majority of cases die before the fourth day. The convulsions may subside and the child later suffer from epilepsy or paralysis. The slight hemorrhage may give rise to no immediate symptoms. Many cases of idiocy, dull mentality, epilepsy, hemiplegia and diplegia, trace their origin to intracranial injuries of parturition.

The diagnosis of meningeal hemorrhage or internal hematoma of the meninges may be made with great accuracy, with the history of an external hematoma, difficult labor and recurring convulsions. The treatment of intracranial hemorrhage is almost hopeless, even if the convulsions are relieved, the residual disabilities are not lessened.

CASES ILLUSTRATING CLINICAL HISTORY OF MENINGEAL HEMORRHAGES.

The following is the history of a case that I think would be diagnosed as *feeble vitality, congenital debility or heart disease.* Baby born after a normal labor lasting two hours, head presentation, died aged six hours, growing blue in the face, no convulsions.

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Autopsy.—Plump baby, thymus large, lungs expanded, ecchymoses in the pleuræ; stomach and intestines contain large quantities of viscid mucus; *brain:* a film of blood over motor areas of cortex, large clot at base of brain about cerebellum and medulla, small clot in ventricles. Here the hemorrhage was due to the moulding of the head in an easy labor.

Meningeal Hemorrhage Causing Convulsions.—Called at 9 P.M. to see a child born sixty-three hours before; mother primipara, twin birth, the fetus undeveloped, $^{8}/_{12}$ gestation, 2 placentas, 1 shrivelled, small; 1 normal. Second stage lasted seventeen hours, presentation right occipito-posterior; instruments used; easy delivery; cord tight about neck. Child slightly asphyxiated; an extensive edema of neck, present for thirty-six hours; a hematoma size of hen's egg on left occipital bone, slight forceps' abrasion on left trapezius; defecation and urination normal; umbilicus, defecation and urination normal; umbilicus healthy; heart sounds strong; respiration normal. The alarming symptoms were languor, cyanosis and convulsions. The first convulsion occurred when the child was fifty hours old. There was a second convulsion in the latter part of the morning, a third in the afternoon, and at 8:30 P.M. a series of severe convulsions. Some of the attacks of eclampsia followed the act of swallowing, showing a very sensitive pharyngeal reflex. Child blue and feeble; evening temperature 103°F.; advised cold to the head and chloral. The child had no more convulsions and died at 11:30 P.M.

Next is the history of a case, with *long continued convulsions, partial recovery, development of epilepsy, death in status epilepticus.* Called to see child seventeenth day of life for frequent convulsions. History: born after normal labor, easy head presentation, non-instrumental; second day of life child began to have convulsions, would become cyanotic, breathe rapidly, and have twitching of extremities. In the intervals of relaxation baby apathetic and would not nurse; fed breast milk. Defecation in order; temperature in axilla for last ten days 102° F. Child had grown weaker and thinner, five to fifteen convulsions a day, generally in afternoon and night. Examination on the seventeenth day: child very thin; temperature 97.6°F.; respiration 50; heart sounds weak, 120; right parietal large hematoma; child grew cyanotic when handled; umbilicus unexcoriated; passed a foul mucous stool. Later history: the convulsive attacks gradually grew less frequent; in later infancy ceased for a month; then re-

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curred at frequent intervals; child was fat with a type of flaccid palsy; blind from optic atrophy; child died aged two years in status epilepticus.

I wish also to report a *case of difficult delivery, child born asphyxiated, completely resuscitated; there were no convulsions; no rigidity or loss of power.* This child throve, and, until it was eighteen months old, there was no evidence of intracranial injury. At this age it was noticed that the child moved awkwardly and could not walk; signs of spastic paralysis developed, and now, at the age of four years, the patient is the victim of spastic diplegia, Little's disease. There are exaggerated reflexes, stiffness, and incoordination in the upper and lower extremities, strabismus and dull mentality. The cause of the child's disability was a meningeal hemorrhage of the cortex, over both motor areas, with secondary degeneration of the anterior and lateral pyramidal tracts of the spinal cord.

ASPHYXIA AND ATELECTASIS PULMONUM.

In Buffalo, in 1902, seventeen deaths were recorded from asphyxia, atelectasis and compression of the cord, being 3.6 per cent. of the total mortality in the first month of life. Asphyxia and cerebral hemorrhages are usually caused by the same conditions of dystocia, and have in common the symptoms of prostration, cyanosis and irregular breathing. Asphyxia is ordinarily completely relieved by artificial respiration, but a number of cases are seen, where treatment is only able to effect a partial expansion of the lungs; the infant lives for days and weeks with one-third to one-fourth of its lungs in working order. It is feeble, has a subnormal temperature, will not cry or nurse, breathes rapidly, irregularly; suffers from frequent cyanotic attacks, in one of which it may die.

In congenital atelectasis there may be an easy labor, no asphyxia, but from inherent weakness or immaturity of the respiratory muscles and nerve centres the baby will not expand its lungs. It leads a cold, cyanotic vegetative existence; refuses food and is apt to die suddenly in an attack of cyanosis. In both asphyxiated infants and those suffering from atelectasis there is feeble or absent breath-sounds at the bases of the lungs. Underweight children or those born asphyxiated should be occasionally ausculated and closely watched. With lividity, rapid or irregular breathing, oxygen, mustard baths, artificial respiration, forced feeding and

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hypodermic stimulation should be used. With vigilance a large number of these cases may be saved.

Both asphyxia and atelectasis are fertile causes of bronchopneumonia.

SEPSIS.

A large proportion of fatalities in newborn infants are from sepsis. The avenues of infection are numerous, and include the eye, ear, nose, mouth, skin, digestive and respiratory tracts, and umbilicus. The germ may be any one of the pathogenic bacteria. The patient's resistance to disease is very slight, probably owing to deficient leukocytosis and defective development of the lymphatic apparatus. When puerperal fever prevailed young infants were frequently infected by their mother's milk. In Prague, after the introduction of antiseptic methods in the hospital, the mortality of the newly born sunk from 30 per cent. to 5 per cent., and the number of feverish children from 45 per cent. to 11.3 per cent. Indeed, the conditions are often more favorable for infant life in a fine hospital than in an ordinary dwelling. According to Birsh-Hirschfeld prenatal infection, save for lues, is almost impossible. The healthy placenta is a perfect filter for germs. It is only when the placenta is diseased or saturated with bacteria that intrauterine infection is possible. It is favored by partial detachment of the placenta or frequent hemorrhages. The greatest source of danger is the amniotic fluid after the bag of waters is prematurely broken. The amniotic fluid is polluted by the examining finger or by an ascending infection. The fluid is aspirated into the lungs or swallowed and infection may become general or localized, in the respiratory or gastroenteric tract. The germ is generally the colon bacillus. A period of incubation may occur, and several hours or a day or two may elapse before symptoms appear.

Whatever be the microbe or portal of invasion, the infection soon becomes general, and nearly every autopsy will reveal a pneumonia. To discuss the matter in detail would be to allude to symptoms arising from lesions of every organ. The general clinical picture is, fever, cyanosis, rapid breathing, convulsions, vomiting and diarrhea. (Mother usually well, free from fever.) It must be remembered that in a prematurely born child, weighing from 1,500 to 2,000 grams, a virulent infection may exist without a rise in temperature. Most cases of sudden death may

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be explained in this way; there is no ability to defend the organism against toxins.

It is usually stated that the umbilicus is the vulnerable point. I doubt if many infant deaths are due to this cause. In Buffalo, in 1902, three deaths of 471 are returned from lesions of the navel, at the fourth, nineteenth and twenty-second day; it is possible the condition is more frequent, owing to the fact that physicians are slow to acknowledge that a preventable disease has occurred in their practice.

Respiratory inflammations are excessively common in the first month. The disease may start from purulent coryza, an imperfectly expanded lung or a pulmonary apoplexy. The infection may come from foul atmosphere, unclean bedding or mattress, as seen in the Heidelburg epidemic, where an epidemic of 8 cases arose from staphylococci in straw beds, but it is more frequently caused during parturition, by the aspiration of amniotic fluid, contaminated by the colon bacillus, streptococcus or staphylococcus. The number of deaths reported, 40, 8.5 per cent., does not accurately state the frequency of the disease, as a majority of infants dying in the first month have a bronchopneumonia. Pneumonia is diagnosed principally by objective signs, fever, cyanosis and rapid breathing. There are no characteristic physical signs in the chest and the treatment is hydrotherapy, counter irritation and stimulation. Delestre states that the majority of autopsies in prematurely born children show a hemorrhagic bronchopneumonia, causing almost an absolute obstruction in the bronchial tubes.

Richter, of Vienna, made 1,525 autopsies upon children dying suddenly in the first month. He found the most frequent cause of death was a capillary bronchitis, which killed by asphyxia. The bronchitis was occasionally associated with bronchopneumonia or intestinal catarrh. As to other causes of sudden death, Epstein said the diagnosis of hypertrophy of the thymus was a term used to mask our ignorance of the real cause of death.

GASTROENTERIC INFECTIONS AT BIRTH.

Gastroenteric infections at birth are occasionally seen and receive small mention in text-books. In Buffalo, in the year ending December 1, 1902, there were among infants under one month, thirty deaths from acute gastroenteric disease, and forty from marasmus and inanition, making 16 per cent. of the total mortality under four weeks.

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With some children a congenitally-imperfect digestion exists, the digestive ferments are weak, the congested gastroenteric tract is coated with mucus, and autointoxication or infection readily occur. Vomiting, diarrhea and colic are easily excited, and may be due to mere dyspepsia, to grave gastrointestinal infection or autointoxication, or to general sepsis. Delestre describes a chronic infection causing infantile atrophy or athrepsia. Dyspepsia may be caused by colostrum breast milk, or by injudicious food mixtures.

Severe gastroenteric infection is in my experience more frequent and fatal in newborn babies than lesions of the umbilical wound or vessels. The germ may enter the digestive tract in various ways, on the *accoucheur,* or nurse's finger in the toilet of the mouth, by breast milk or food-prescription. It may be sucked from the breasts of the mother or may be introduced by the swallowing of polluted amniotic fluid or vaginal secretion during the infant's sojourn in the parturient canal. Amniotic fluid is often present in the stomach of the newborn babies, and is, of course, not always pathogenic. Karlinski discovered pus cocci in the milk of a sick mother and in the food and intestinal contents of her baby, dead of septicemia. Before the days of aseptic midwifery, a close association was observed between infantile diarrhea and child-bed fever. The mother was infected during or after parturition and the baby swallowed bacteria in the breast milk. Nowadays, the mother remains well, while between 9 per cent. and 10 per cent. of all children born die in their first month of life.

Between dyspepsia and gastroenteric sepsis, the dividing line is vague, often depending on the dose and virulence of the poison and the resistance of the individual. Microbes habitually inoffensive may become virulently pathogenic.

In Grosz's statistics of the morbundity of young infants, of 447 newly born hospital children, 254 or 57 per cent., showed food vomiting or dyspeptic diarrheas. He says the rapidly fatal choleriform diarrheas, formerly seen, now seldom occur.

Grosz's diagnosis of dyspepsia was based on an illness of ten days with vomiting and frequent mucous stools. He describes three grades of dyspepsia: (1) Several yellowish green stools daily; (2) same symptoms with vomiting; (3) stools dark, green color, frequent vomiting and a rapid loss in weight.

The 5 cases of diarrheal disease in newborn children which I report are of a much graver character than Grosz's subdivisions

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of dyspepsia. They are best described as gastrointestinal sepsis or ileocolitis. The babies were of good weight and sound, vigorous physique. They were evidently poisoned during or soon after birth by some virulent germ which, besides exciting acute gastroenteric irritation, profoundly depressed the respiratory and cardiac centres. The three survivors showed no signs of digestive or constitutional weakness in later infancy. As soon as the poison had been eliminated or had lost its virulency the children throve with their mother's milk.

First Case.—This is possibly one of *severe dyspeptic diarrhea,* yet the baby was failing so rapidly in the hot month of August that the illness was diagnosed as an ileocolitis. The confinement was normal and the umbilical wound healed quickly. The mother had plenty of milk and attempted to nurse her baby boy; a diarrhea quickly ensued with six to eight green, mucous stools a day. Temperature, 100°-103° F. The breast milk was analyzed and proved to be a colostrum milk, highly albuminous. In spite of treatment and a change in diet to cereal-gruels and whey the diarrhea continued and increased in severity for twelve days. At the fourteenth day of life the frequency of the stools, the child's prostration and emaciation (one-fifth of the weight, twenty ounces, had been lost) made the patient's condition somewhat alarming. A wet-nurse was procured with a gradual amelioration of all symptoms. Later, the mother nursed her child, and at the age of four months it weighed twenty pounds.

Second Case.—The patient was the third child, born October 15th; two other children died shortly after birth. The baby was born with the cord about the neck; the baby was partly asphyxiated and well resuscitated. Immediately after its birth the child seemed feverish, apathetic, and would not nurse its mother or a wet-nurse. For the next three days the baby appeared very ill; October 16th, the temperature was 104°F.; on October 17th it was 102°F.; on October 18th, 102°F. It refused the breast, was fed on diluted breast milk, and had two to three foul, mucous stools daily. I first saw the case October 19th, the fifth day of life; the baby was large, well developed, but languid and exhausted. Temperature, 100°F.; pulse, 140. I removed the cord; the umbilical wound was healthy. There was a livid erythema of the mouth and numerous stools of mucus and food débris.

October 20th, same condition of prostration and diarrhea.

October 21st, the patient grew rapidly worse; temperature

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101.5° F.; respiration, 84; pulse, 140. Persistent vomiting of food and bloody mucus, frequent stools of mucus and blood; the baby was almost continually rigid, with opisthotonus and carpopedal spasms.

October 22d-23d, the same symptoms of tonic spasms of the trunk and extremities with frequent vomiting and stools of bloody mucus.

October 24th, the child died at 9:30 A.M.

Autopsy.—The body was much discolored; the lungs were congested at the bases; the right ventricle was distended, esophagus congested; stomach dilated and congested; entire intestinal tract congested and coated with bloody mucus; umbilical vessels healthy; brain not examined.

The duration of the malady was ten days; the patient seemed ill from the moment of birth. In its sojourn (asphyxiated condition) in the parturient canal the baby in efforts at breathing may have swallowed polluted amniotic fluid or vaginal secretion, producing a fatal gastroenteric infection. The nervous symptoms, opisthotonus and carpopedal spasms, were probably the result of general sepsis.

Third Case.—Baby born October 8, 1901. Normal labor, no asphyxia, weight about eight pounds; child nursed on the second day; the breast milk was noticed to be unusually thick and creamy. The baby throve until the fourth day of life; then grew somnolent and would not nurse; the stools became frequent, seven to eight daily, were black, foul-smelling and contained strings of mucus. I saw the patient the sixth day of life. The baby looked very badly, temperature 102° F.; heart, weak and rapid; umbilicus, healthy; a fragment of mummified cord sprinkled with boric acid was hanging to the navel.

From October 14th-19th, the symptoms were, numerous fetid, mucous stools, fever and increasing prostration. The baby was sparingly fed on diluted breast milk, whey and gruels. It was given calomel, intestinal irrigations, strychnia, whiskey and artificial serum.

October 20th, the patient grew rapidly worse; the rapid breathing suggested a pneumonia; the diarrhea continued with frequent attacks of cyanosis. Death occurred October 21st. No autopsy was permitted. The illness lasted ten days. The gastroenteritic tract was probably infected through the breast milk, which was thick and slimy.

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Fourth Case.—The mother was a primipara of thirty. The labor was long and difficult, lasting fifty-seven hours on account of rigidity of the cervix. Opium and hyoscyamus was given in the earlier stages of parturition. Head presentation, delivery was effected by forceps under chloroform narcosis. The *accoucheur* introduced his fingers into the rectum to assist delivery, rinsed them off in sublimate solution, and wiped out the baby's mouth with sterile gauze. The child weighed eight and three-quarter pounds, was at once put to the breast, although the secretion of milk was not established until the fifth day. When twenty-four hours old the baby began to vomit and pass black liquid stools, containing mucus and blood.

April 10th.—I first saw the child on the third day of life; it looked very ill, had cried for hours in a hoarse, gasping way; there was no cyanosis; temperature 103°F., heart so rapid and feeble that the child seemed moribund. With stimulation and intestinal irrigation the child revived somewhat, yet for four days it was very feeble and required heroic doses of whiskey, strychnia and hypodermoclysis. The temperature ranged from 102°-106°F.; respiration, 50-60. There was occasional vomiting and numerous stools, ten to twelve daily, of bloody mucus and food débris; the food was diluted breast milk from a wet-nurse.

After April 13th the baby slowly improved, and the fever gradually subsided. The acute symptoms lasted for six days. Prostration and imperfect digestion continued for three weeks. The mother nursed the child when two weeks old; the baby is now in superb condition.

Infection in this case may have occurred from swallowing amniotic fluid, or more probably from the toilet of the mouth through the finger of the *accoucheur* which had previously been introduced into the mother's rectum.

Fifth Case.—The mother a primipara of thirty-three.

Labor easy, noninstrumental. Baby born September 26th; weight, seven and one-half pounds; the baby was put to the breast and had normal meconium stools for two days. The third day the child showed fever 102°F.; the respiration became rapid and irregular, 80; a serious diarrhea appeared, 12 stools containing blood and mucus in twenty-four hours. I saw the child at 5 P.M.; it had been stimulated with strychnia and atropia. Temperature, 99.5°F.; pulse, 120; color, good. The malady had strangely affected the rhythm of respiration. The breathing was rapid, ir-

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regular and superficial, only an occasional inspiration would inflate the lower lobes; umbilicus, healthy. On the fourth day the temperature at 2:30 A.M. was 101°F.; there were twelve bloody, mucous stools in twenty-four hours; at 9 A.M., the child sank into desperate collapse with marked respiratory failure; after two hours' work with oxygen and hypodermic stimulation, the child improved and at noon was in fairly good condition.

During the next three days, October 1st, 2d and 3d, there was no fever; the respiration was irregular and superficial; there were five to eight black, bloody, mucous stools a day. The baby was so weak that it was fed with a medicine dropper.

On October 4th, the mother began to nurse the baby; the respiration gradually grew deeper and more regular. Mucus and undigested food débris were present in the stools for two weeks; the loss in weight during the first week amounted to only twelve ounces. The baby is six months old, now in good health. The cause of the infection was impossible to discover.

This little series of cases of diarrheal disease in newborn children was seen in consultation practise, among well-to-do families, with good hygienic surroundings. The mode of infection is obscure, but I have a most positive impression that in 2 cases (1 and 3) the mother's milk was the cause of illness and that in 1 case (2) the child was infected in the parturient canal, probably by swallowing amniotic fluid containing the colon bacillus. All of the mothers attempted to nurse their children, and no other food than breast milk was given until fever, vomiting and diarrhea appeared. Colostrum or infected breast milk may excite a severe or fatal diarrhea. If the mother's milk be suspected, nursing should be stopped at once. It should be remembered that while colostrum milk may temporarily be very irritating, that the excess of albuminoid is apt to disappear in a few days, when nursing may be resumed.

The prevention of gastroenteric disease in the newborn is effected by vigorous asepsis, and especially by avoiding unnecessary toilet of the mouth with unsterilized fingers or gauze, as these infants are very prone to infections of the lungs and digestive tracts.

If the physician wishes to succeed in saving a newborn child who shows signs of grave infection, he must keep it under close personal observation. At this early age attacks of sudden cardiac and respiratory failure are frequent and unexpected and require

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prompt hypodermic stimulation with strychnia, atropia and normal salt solution. For food, diluted breast milk should be given. Other adjuvants of treatment are: hydrotherapy, laxatives and gastric and intestinal lavage.

The diseases of the newborn are little studied or understood. The high mortality of the first month could be much lessened if the unnecessary use of the forceps were avoided and surgical cleanliness were observed about the child as well as the mother.

DISCUSSION.

DR. KOPLIK.—I do not see the necessity in maternity hospitals for cleansing the mouths of the infants. The newborn infant's mouth is clean, and if you do not introduce anything infectious into it you will probably not cause an infection. Therefore, in a maternity hospital the principal point should be to cleanse the mother's breast and to keep the nipples thoroughly clean before, and during the intervals of, nursing.

At the City Hospital, some years ago, I was asked to see a number of children who were supposed to have been born with socalled ulcers in the mouth. When I examined these newborn babies I saw that they had what is familiar to all of us as Bednar's aphthæ, and these in some cases were covered with a yellowish pseudomembrane. Upon inquiry it seemed that these children developed these aphthæ immediately *after birth* and in some cases there was a spread of the ulceration in the mouth. I begged the physician in charge to stop washing the mouths of the babies, and as if by magic this socalled epidemic disappeared. I do not know what they do there to-day, but at that time they began the process of keeping the breast nipple clean and leaving the mouth of the infant absolutely alone. The credit of this method of managing the mouth belongs to Epstein, of Prague, who has carried it out successfully for years in the maternity hospital of that city.

DR. ROTCH.—Just before I came away I saw a child which impressed this fact upon me very forcibly. A perfectly normal mother gave birth to a perfectly normal child. She had successfully nursed three other children. The child for three days remained well; the movements were normal. At the end of the week I was called to see the child, who had some intestinal trouble. The movements were of a greenish color and contained some mucus; there was some pain, and on inquiring into the cause of the trouble I found a fissured nipple with a little pus in it. It was carefully treated and cleansed, and immediately the diarrhea disappeared. I do not think there could have been any question in this case but that the fissured nipple with its bacterial

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contents was the direct etiologic factor in causing the intestinal trouble in the newborn. I have observed this in other cases. Nothing else was done in the treatment of this child than the cleansing of the nipple, and it rapidly recovered.

DR. SHAW.—I should like to speak of a rather interesting case I had last summer. I was called to see a baby on the twelfth day after birth. The mother had one of the best nurses in Albany. All went well until the eighth or ninth day, when the child passed a good deal of mucus. The physician gave some castor oil and the trouble cleared up, but the baby began to fail and became much emaciated. The child had two or three slight convulsions. The next day it died, and we were permitted to have an autopsy. Macroscopically, there was nothing found at all; there was no congestion of the lungs; no inflammation of any of the serous or mucous membranes, and there was nothing, so far as we could see, to account for the condition. A bacteriologic examination was made, and the findings are of especial interest after Dr. Hamill's paper. In the blood we found staphylococcus pyogenes albus; in the lungs streptococci and staphylococcus pyogenes albus; in the intestines we found coli communis, and in the pus around the umbilical cord we found bacillus proteus vulgaris and an unidentified coccus.

It seems to me that the subject is of very great importance, and I agree with Dr. Hamill that the classification is rather ambiguous, being based on the symptomatology, while the underlying cause is similar in a great many of these socalled infections of the newborn.

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DISTURBANCES OF RESPIRATION IN THE NEWBORN.

BY W. REYNOLDS WILSON, M.D.,
Philadelphia.

The form of respiratory disturbance which first confronts the practitioner in his attentions to the newborn is asphyxia. Closely associated with this is congenital atelectasis. These two conditions may be included under the general heading of respiratory failure. As subjects for clinical study, outside of their obstetrical significance, they are unimportant, while atelectasis, associated with other lesions and in later newborn existence, is not necessarily marked by respiratory failure alone.

It is the suspension of respiration arising from special causes which may claim our attention, although the enumeration of these causes is not the object of this paper. It is rather that the study of individual symptoms may emphasize the relationship of the symptoms themselves to the underlying condition.

As to the special causes of failure of respiration, deficient action of the abdominal muscles is frequently observed, especially in asphyxia, but it has a special significance in premature infants in that it limits respiration and interferes with the supply of oxygen, reducing to this extent the infant's nutrition. Failure of respiration from traumatic causes has been observed, the result of pressure in intracranial hemorrhage and the result of paralysis of the abdominal muscles due to spinal injury during birth. (A. H. Davisson, *Philadelphia Medical Journal,* Vol. ii., No. 8.)

Irving M. Snow (Transactions of the American Pediatric Society, Vol. xiii., p. 123), recites two instances of recurring respiratory failure, apparently of bulbar origin. He advances the opinion that the condition is dependent upon a transitory and powerful influence upon the medulla, the result of infection of some sort affecting possibly the inhibitory centres. The bulbar theory of the malady presents itself, in his opinion, on account of the existence of morbidly active pharyngeal reflexes, which existed in both cases. Two similar cases which he recites (1) that of Dr. George Keith, the *Lancet,* November 24, 1900; (2) that of Dr. Westcott, ARCHIVES OF PEDIATRICS, October, 1897, point

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to the possibility of an infectious influence exerted upon the nervous system. An accidental surgical infection probably existed in the first of these cases, that of Dr. Keith, in which attacks of failure of respiration, more or less prolonged and associated with cyanosis and rise in temperature, came on within a few hours after circumcision. In the second case, that of Dr. Westcott, the child, slightly beyond the newborn period, was seized in the course of influenza with an attack of superficial breathing culminating in absolute stopping in respiration and associated with excitability of pharyngeal reflex similar to that observed in Dr. Snow's case. In this case, therefore, the infectious character of the symptoms was also undoubted.

In contradistinction to this class of cases are those instances of passing respiratory failure in the newborn due to toxic influences, the result of alimentary disturbances. The evidences of reflex irritation are more or less pronounced in degree, shown for instances in some cases by prolonged attacks of drowsiness, which are apt to overcome even breast-fed infants and which are promptly relieved by purgation; or, in other instances, by sudden nausea, which shows itself in the form of syncope with respiratory failure.

It is not difficult to find in every-day practice instances of the latter condition. The following is typical:

Infant of Mrs. B. A well-nourished, vigorous child showing early restlessness, apparently from hunger, on the fifth day while on the nurse's lap became suddenly pale and ceased to breathe. The attack lasted for the fraction of a minute and was accompanied by slight cyanosis and coldness of the extremities. There was no spasm. The infant was revived by a warm bath. A similar attack occurred on the following day. The temperature had risen on the third day to 101½°F., returning to normal on the eighth day. The child was put upon minute doses of calomel and the condition was relieved. Improper nourishment was no doubt the cause of the disturbance. A weak solution of condensed milk had been given to satisfy the child up to the beginning of lactation, the latter having been delayed beyond the usual period. From the time at which the discharge of meconium had ceased until lactation the stools had been unduly frequent and greenish in character.

A rise in temperature associated with the symptoms just described may show itself in advance of the digestive disturbance;

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and, in some instances, may be mistaken for the fever of inanition described by Holt. Indeed, it is not infrequent for an apparently healthy infant, fed at the breast, to undergo in the first few days, before lactation is fully established, digestive disturbance which is shown in mild pyrexia, the course of such a feverish attack ending in colic and diarrhea. This is especially the case in infants of primiparæ.

Instances of acetanilid poisoning from application to the umbilical wound and external surface reported by Snow (Transactions of the American Pediatric Society, Vol. ix., p. 39) may be included among the causes of respiratory failure, although the most pronounced evidence of poisoning in such cases lies in the cyanosis, the respiratory failure being merely an accompaniment of collapse.

DYSPNEA.—This may arise from certain congenital conditions. Cardiac malformations come first in order. These are accompanied by a deviation of the blood current from its entrance to the pulmonary circulation. For this reason the condition is marked by cyanosis. Although the latter is always present the degree of dyspnea may vary. The dyspnea is constant in character, especially if the malformation be extreme; that is, other than the persistence of late fetal conditions, such as patulous foramen or ductus arteriosus.

The dyspnea of pneumonia is marked by the absence of paroxysmal attacks. Frequently the breathing is irregular and takes on the character of Cheyne-Stokes respiration. The want of oxygen, added to the toxemia, rapidly induces somnolence and apathy. In congenital atelectasis the cyanosis is marked from the beginning, partly for the reason that the stagnant pulmonary circulation interferes with the closure of the foramen ovale, the systemic circulation being charged with venous blood which finds its way directly into the left heart (Henoch). The absence of fever and râles, together with the presence of dullness over the area of collapse, if the latter is extensive, are distinguishing features in atelectasis. In premature and weakly children with pneumonia fever may be absent. The physical signs may also be deficient, so that the differential diagnosis between pneumonia and atelectasis may be impossible.

Stenotic dyspnea in the newborn presents an interesting field of study. Congenital stenosis of the larynx may occur in the form of a contraction of the aryepiglottic folds (O'Dwyer, Trans-

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actions of the American Pediatric Society, Vol. ix., p. 180). The rima glottidis is not involved, so that the voice is unaltered. Spasmodic respiration is the prevailing symptom. Accepting the fact that simple spasm may occur it is yet probable that the labored crowing inspiration sometimes observed may, in certain instances, be due to such malformation. Spasm may be an accompaniment of a form of membranous inflammation of the throat due to streptococcus infection, observed by J. Lewis Smith and described by Holt (Diseases of Infancy and Childhood, second edition, 1902).

Dyspnea due to enlargement of the thymus gland.—The term "enlargement" in this connection is indefinite; in the newborn there exists a variability in the size of the gland, true hypertrophy being found usually in the latter months and existing as a congenital condition only in cachectic infants in whom a hyperplasia of certain lymphatic tissues, markedly the bronchial glands, is also found (Grancher, Marfan et Comby, *Mal. de l'Enf.*, Paris, 1897). The attacks of dyspnea in such infants are purely convulsive and are usually not due to direct pressure. Reflex irritation due to functional enlargement of the gland is illustrated in a case reported by Kurzem (*Arch. f. Kinderhlknde.*, Vol. xxvi., p. 277) in which a dyspneic attack was evidently of thymic origin, as he was able to find by clinical examination a functional enlargement of the gland. The symptoms disappeared entirely after the use of ice applications. It should be recalled that the apnea which accompanies enlargement of the thymus may show itself with great suddenness and may terminate fatally. For this reason in an instance of sudden death in the newborn the possibility of enlargement of the gland should be entertained as bearing upon the cause of death, inasmuch as the respiratory disturbance may have been of such short duration and of such sudden development as to have escaped notice. Beyond this the thymic origin of early dyspnea is not of importance.

Before dismissing the subject of dyspnea arising from disturbances in the cardio pulmonic system the differentiation of broncho pulmonary hemorrhage from other thoracic conditions may be properly considered. Pulmonary extravasation has, together with pneumonia and atelectasis (the two pathological conditions more frequently met with), the common etiological factor of depressed vitality, which belongs to premature and cachectic infants; according to Demelin (Grancher, Marfan et Comby, *Traité des Mal. de l'Enf.*, Paris, 1897) the initial weight of infants

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under his observation suffering from pulmonary apoplexy was from 1,800 to 2,200 grams. Dullness and a diminution in the vesicular murmur may be common to all three affections; in pulmonary hemorrhage, on the other hand, the dyspnea may be replaced by feeble respiration, with but only occasional paroxysms of difficult breathing. The cyanosis in this condition is likely to be more pronounced. The temperature is, as a rule, subnormal. Râles are usually present. The actual observance of extravasation from the mouth is conclusive. The history of obstetrical trauma or the possibility of an associated infection may have a bearing upon the etiology of hemorrhage. Cough as a symptom, owing to the absence of bronchitis, is absent. Subcutaneous extravasations are frequently seen as an accompaniment.

Dyspeptic dyspnea.—Where the signs of intrathoracic trouble are wanting the symptoms may point to alimentary disturbance. The dyspnea is toxic in origin and has nothing to do with intra-abdominal pressure. The dyspnea is usually not the primary manifestation, in most instances, of indigestion; green stools, scanty urination, fever and anorexia are likely to precede the attack. The difficulty in breathing is accompanied by cyanosis and rapidity in the pulse rate with diminution in volume. Emaciation in these cases usually precedes the appearance of the dyspneic attacks; and, taken with the intestinal irritation, point to alimentary disturbances as the underlying condition. It would hardly be in accordance with the description of Henoch to classify these cases in the newborn under the heading of "dyspeptic asthma." The latter condition is one in which the asthmatic attacks appear as the initial and primary symptom of arrested digestion. In their typical form they appear in older children.

Diaphragmatic hernia offers a complexity of thoracic and abdominal symptoms among which dyspnea predominates. The difficulties in diagnosis in this malformation are illustrated in a case described by Booker (Transactions of the American Pediatric Society, Vol. ix., p. 100). The rarity of this condition on the one hand, and the frequency with which intestinal disturbance is met with, on the other, make it probable that few observers have had the opportunity of confusing the symptoms. In going over the interesting report of Dr. Booker's case the most striking point of interest was the accession, after periods of comparative comfort, of distressing dyspnea, which was accompanied by pain and followed by collapse. In this case the difficulty in breathing was

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at first attributed to the pressure of gas in the stomach and bowels with the probable absorption of injurious substances from the intestine. The following quotation describes the thoracic condition: "The respirations were sixty to eighty to the minute during the attack and there was recession of the soft parts of the chest with inspiration. Vesicular breathing could be heard over the whole of the chest but it was much more feeble than normal. The heart sounds were greatly obscured by the frequent and violent movements of the chest, but it could be safely determined that no murmur existed. The abdomen was soft and not distended, although there was a considerable escape of gas from the intestines. Pulse 130 to 160. Percussion resonance over the chest was accentuated and the tympanitic sound of the abdomen was transmitted high in the thorax."

Dr. Booker writes that he has been able to find but one case in which the diagnosis was made during life and confirmed at the autopsy. This was an instance observed by Ahlfeld, in a child in the second period of childhood, who became habitually cyanotic from the most trivial cause, as, for instance, the shock of being put in the bath. The heart could be located in the right axilla, the left thoracic region giving an intestinal note on percussion.

Rapid respiration.—Rapidity in respiration, in contradistinction to dyspnea, is commonly met with. It may be paroxysmal and transient or continuous. Dyspeptic and febrile conditions may be the underlying causes. It may be a symptom of the common pulmonary affections, as bronchitis and pneumonia. In systemic septic infection rapidity in respiration is one of the accompaniments of fever, which is always present. As a symptom, however, it may be out of all proportion in value to the other symptoms. Yet it may be the first indication of disturbance. The symptom may be misleading relative to thoracic conditions. It requires, therefore, careful clinical investigation to place it in its proper position. The following case is reported in order to illustrate the linking of respiratory disturbance, as a symptom, to septic intoxication:

An infant, born August, 1902, showed on the evening of the third day a sudden rise in temperature amounting to 103°F. The fever had been preceded by restlessness and was accompanied by rapidity in respiration. The latter was from 60 to 80. The breathing was abdominal in character, and exceedingly shallow. Auscultation and percussion gave no evidence of intrathoracic trouble.

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There was turgescence of the veins of the head and the venules over the chest and abdomen with general mottling of the skin. Lactation had been established early and the child had begun to nurse regularly. There had been slight icterus but no alimentary disturbance. The cord was well mummified at this date and the umbilical fold showed no irritation. The rapidity in respiration subsided with the fall in temperature, which occurred on the fifth day. On the sixth day the beginning of a virulent ophthalmia was noticed.

The treatment followed was the use of the ice-cap and the administration of the fifth of a grain of calomel divided into six doses.

The mother, whose condition, from the following description, bore directly upon the ophthalmia in the newborn, had shown anemia and loss of appetite during the last days of her pregnancy. She continued, during the early days of her puerperium, to show evidences of septic intoxication, which, however, amounted only to slight nervous agitation and anorexia. There was no rise in temperature beyond a slight elevation from engorgement of the breasts. On the tenth day she suffered from a pronounced rigor followed by fever. In irrigating the uterine cavity it was discovered to be the seat of a purulent endometritis. The patient developed a pyemia with irregular exacerbations of fever and fresh metastatic foci. Finally it became necessary to excise the Fallopian tubes, which were the seat of an acute purulent salpingitis. The patient recovered after a protracted illness. The blenorrhea in the child subsided in the course of six weeks.

It would be more striking than exact to connect the respiratory disturbance in this case with the beginning ophthalmia. The fact is in reality that the septic intoxication in the child was probably the result of a mixed infection, just as the septicopyemic condition in the mother was of such origin, since the virus of the gonococcus, as is well known, is prone to set up localized suppurative foci in contradistinction to general septic intoxication (Fishl, *Sammlung Klin. Vortrage*. 1897-8, No. 220). Blenorrhea in itself would, therefore, hardly account for the toxic disturbance which gave rise to respiratory changes observed in conjunction with other symptoms. The source of the septic infection could scarcely be considered of prenatal origin, that is, as a direct maternal infection in utero, as septic infection in the newborn of this origin is accompanied by marked blood dyscrasia and de-

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generative changes which rapidly end fatally (Miller, *Jahrb. f. Kindhlke.*, 1888, No. 28, p. 155).

The source of the infection must, therefore, have been obstetric rather than congenital, and may have arisen from entrance of the infecting elements through the mouth or umbilical fold, or by an invasion of the bronchial system. The amniotic sac had been intact until the completion of the second stage of labor, so that the opportunity for invasion by the strepto- or staphylococci, present in all probability in the genitourinary tract before the birth of the child, occurred during the passage of the birth canal after rupture of the membranes.

The penetration of the child's alimentary tract by bacteria in the milk during the early stage of the puerperium is hardly a likely explanation of the child's condition.

DISCUSSION.

DR. SAUNDERS.—One cause of disturbance in respiration which exists in our cities is gas. The houses are now mostly heated by steam radiators, supplemented by the gas log. The products of combustion are thrown off into the room. One case which I had was that of a newborn child with acquired atelectasis, which required my presence, or that of my assistant, for twenty-four hours. This is a real menace to the safety of newborn children. In pure apnea there is nothing, I believe, so efficacious as flagellation. I usually tell the attendant to take a little rubber band and flip the soles of the feet whenever the child begins to get tired of breathing. Of course, I use medicinal means also. In cases of apnea where there is a necessity of using an aspirating syringe, I find they can be dealt with most effectually by causing the child to gag by putting an aseptic finger into the pharynx. You will be astonished sometimes to find that, in a child with interrupted breathing, who is getting worse and worse, with a number of moist râles in both sides of the lungs, the lungs clear up and regular breathing is established.

DR. HAMILL.—I have been very much interested in this paper for two reasons. First, because I have known for a long time of the valuable work which Dr. Wilson has been doing in the study of the diseases of the newborn, and, in the second place, on account of the close relationship of this subject to that of my paper of yesterday.

In the large majority of cases of infection of the newborn, some degree of respiratory embarrassment exists; indeed, it is such a common manifestation in these cases that its presence al-

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ways suggests to me the likelihood of an infectious condition, and I believe that, in most instances, it is an indication of an infection or an intoxication. Many cases of socalled inanition fever, ieferred to by Dr. Wilson, occur in the course of epidemics of the infectious conditions of the newborn. I have been inclined to consider these instances of mild infection.

In Dr. Snow's paper, he stated that in one of the maternity hospitals in Prague, before the days of rigid antisepsis, 44 per cent. of all infants born showed some elevation of temperature during the first weeks of life. After the introduction of antiseptic methods, this figure dropped to 11 per cent. This is quite suggestive, and confirms one in the belief that these fevers are infectious in character.

DR. WILSON.—Since writing this paper I have had a case of pneumonia with collapse of the lung resulting from infection during birth. The child was injured by the application of forceps, the region at the junction of the neck and shoulder on the right side was the seat of abrasion; suppuration and ulceration, resulting in a small abscess of the subintegumentary tissues, appeared on the eighth day, with a slight rise of temperature. As soon as the abscess was opened the child recovered. Three or four days after that he lost his appetite and again had a slight fever with collapse of the lower lobe of the right lung, due probably to the depressing effects of the early septic intoxication.

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PRINCIPLES OF INFANT FEEDING AS BASED ON THE EVOLUTION OF MAMMALS.

BY HENRY DWIGHT CHAPIN, M.D.,

New York.

The satisfactory and systematic consideration of the subject of substitute infant feeding is largely dependent upon pediatricians agreeing on some basic principles, and these can only be established by patient study of biology.

It is fallacious to state that infants must have milk because all young animals are carnivorous, as no young mammal is carnivorous, that is a flesh eater, as far as I can find. It is meaningless to state that infant's food should be milk because milk contains animal proteid, for meat and fish are also animal proteid, but not suitable for infant feeding. It is likewise an error to teach that the differences between human milk and cow's milk lie solely in the varying percentages of casein or caseinogen and lactalbumin. I have been having the proteids of milk separated before my classes and every time I have seen it done the more convinced I have become that the proteids of the two milks differ radically. In the first place, the caseins are so unlike in their behavior with reagents that there is no comparison between them, and after separating the casein and the albumin, considerable quantities of albumoses and peptones are found, particularly in human milk, these forming about one-third of the total proteids of human milk. In some recent analyses of human milk, fully one-third of the proteids, consisting of albumoses and peptones, have been classed as sugar, owing to the faulty methods of estimating proteids employed. When it comes to curding milk with rennet and a trace of dilute hydrochloric acid, the curds formed in human milk and cow's milk are so unlike that even the nurses who look on are struck with the differences.

I do not think it is too much to say that any one who has worked with milk itself, and not with reports of milk analyses, would hardly give much weight to the statement that the differences between human milk and cow's milk lie only in the dif-

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ferent percentages of casein or caseinogen and socalled lactalbumin present. Any doubter should work with milk after modern methods of analysis, and he would soon be convinced that the old teaching is erroneous. He would soon see that the caseins have very different properties. This may be due to their combination with different salts, but I am speaking of the caseins as we meet them and deal with them, and not of their ultimate composition.

There are certain principles in dietetics that are fairly well established. Among these are, that a certain minimum quantity of digestible proteid is needed by each animal, and also certain quantities of fat and carbohydrates to supply energy and heat, which quantities depend largely on the expenditure of energy and heat by the animal. It has further been established that a certain amount of ballast, or bulky material, is needed to properly occupy and distend the digestive tract, and that best results attend giving food naturally adapted to the particular species. If any one thing has been established in artificial feeding of young animals, especially of infants, it is the great advantage of feeding the food naturally adapted to the young animal, viz., mother's milk.

When an analysis of milk is made it is found to contain the five main food elements—fat, proteids, carbohydrates, mineral matter, and water. All milks agree in this respect, but the percentage of composition varies greatly. The vital tissues of the body are essentially proteid and we know that proteids cannot be produced by animals from fat or carbohydrates. We would, therefore, naturally expect to find rapid growth in the young animal corresponding to high proteid in the mother's milk, and this is exactly what we do find. After eliminating differences of percentages which show potential food value, we find that milks differ radically in the character of curds formed. The curds are formed from portions of the proteids, so the essential difference between milks lies with the proteids and practically is more of form than of ultimate composition.

A razor and an axe might be analyzed and both found to be composed of iron and carbon in slightly different proportions. Adjusting the proportions of iron and carbon would not make a razor of an axe. Both are to be used in cutting, but they are intended to cut different substances, and their difference in form has this object in view. Now a study of the proteids of milk

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shows them to be of nearly the same composition, but they assume different forms, as, I believe, for specific purposes. It is beyond our power to make proteid, but we can study the purpose of the different forms the proteids of milk take under the action of the digestive juices and act on the information so obtained. Much light will be thrown on this subject by a study of the evolution of the mammary function.

FIG. I.—MAMMARY GLAND OF EARLY MAMMALS. FROM GEGENBAUR.

Away back in the past there were no mammals such as we see around us now. The female laid eggs and incubated them as birds do.[1] The young which were hatched in an undeveloped state were left in a nest and were nourished by milk ejected from the mammary glands of the mother into the mouth of the young animal, which had a peculiar shape that enabled it to cover the numerous outlets of the glands, as no teats were present. (See Fig. I.)

In a higher form[1] (see Fig. II.) the egg, after being laid, was deposited in a pouch or mammary depression in the abdomen of the mother and the milk was ejected along certain tufts of hair, from which the young animal obtained its nourishment, no teats being present in this form.

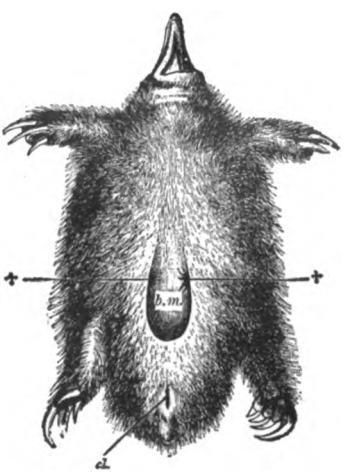

FIG. II.—POUCH WHERE EGG IS INCUBATED. FROM WIEDERSHEIM.

In a still higher form (see Fig. III.) the egg was incubated inside the body, and the young animal, immediately after birth, was attached to a teat in the abdominal pouch of the mother, by the mother, where it grew fast to the teat, and became as much attached to the mother as if it was attached by the navel cord to the placenta.[1] (See Fig. IV.) Such a young animal is called a mammary fetus and is nourished by milk ejected by the mother into its mouth,

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it being unable to suck. Most animals of these classes have become extinct. In the kangaroo there is a uterine development of about thirty-eight days, and a mammary development of eight months. The mother attains a height of seven feet and the young at birth is no larger than the young of a mouse, but on the teat attains to a weight of about ten pounds.[2] At birth it has only a trace of a brain, but its lungs, heart, and kidneys are perfectly formed. Its digestive tract is rudimentary.

FIG. III.—PREPLACENTAL NUTRITION AND DEVELOPMENT. FROM DALTON.

In the next higher form (see Fig. V.) we find that after the ovum has developed to a certain extent, instead of leaving the uterine cavity and becoming attached to a teat, that it becomes attached to the uterine wall and has a period of placental development. In America only one form of implacental animal, the opossum, is known. In Australia, where evolution has not progressed as far as in other countries, there are many species of implacental animals.

FIG. IV. — MAMMARY FETUS OF KANGAROO—NO PLACENTAL STAGE. FROM PARKER AND HASWELL.

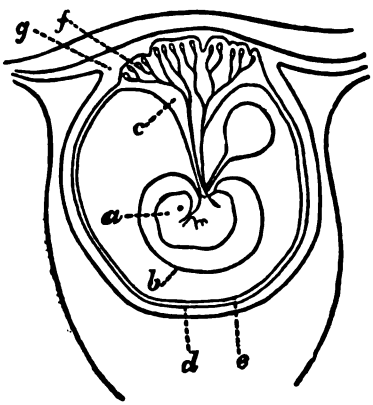

FIG. V.—PLACENTAL NUTRITION. FROM CADIAT.

Placental animals show wide differences in state of development at birth. We see some born blind and helpless—puppies and kittens—others with fully developed nervous systems but poorly developed digestive systems—calves, lambs, colts—and others almost perfectly developed in every respect, of which the guinea-pig, which is said to be able to eat with its mother the day after birth, is an example.[3]

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These varying states of development at birth seem to be closely connected with the mother's means of self defence. The young of carnivorous animals which are not preyed upon, are brought forth in a very poorly developed state because the parent is able to protect them. The young of herbivorous animals, whose safety depends on keen senses and flight, are brought forth with fully developed nervous systems and are able to follow the mother as soon as born. The guinea-pig represents the type of inoffensive and defenceless animals whose existence as a species depends on being reproduced in great numbers, which are soon able to care for themselves.

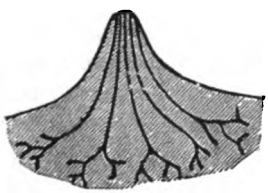

FIG. VI.—DIAGRAM OF HUMAN NIPPLE. FROM GEGENBAUR.

Comparative anatomists tell us that in the early stages of development, embryos of all kinds of animals are so much alike that it is almost impossible to tell them apart, and that the embryos of our present mammals in the course of development show traces of the development of a marsupium or pouch like that of the kangaroo.[4] In the human mother there is indelibly stamped the record of a time when there was no placental connection with the fetus, for just about the time the placenta is formed (third month) the mammary glands begin to secrete colostrum. This time corresponds to the period at which birth takes place in implacentals or marsupials and the fetus becomes attached to the teat. The human mammary gland also shows vestiges of the state in which there was no nipple, the gland having many outlets, as in the lower forms of mammals. (See Figs. VI. and VII. Compare with Fig. I.)

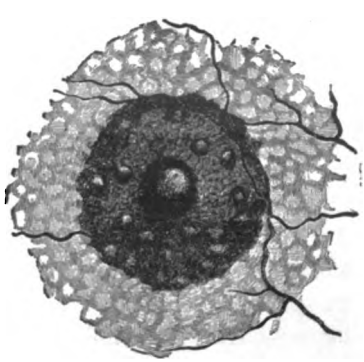

FIG. VII.—HUMAN NIPPLE. FROM EWETT.

The digestive tract in the embryo, as is well known, is at first a straight tube, which gradually develops into the same form as that of the mother. In the kangaroo this change takes place at the teat and the stomach changes from a simple pouch to a sacculated

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organ. No one would think the artificial feeding of an animal whose mouth grew fast to a teat, and who was nourished without any effort on its part by the mother injecting the milk into a gullet, specially arranged to prevent the milk from passing into the lungs, was only a matter of supplying fat, proteids, carbohydrates, etc. It would at once be said that the young animal and the mother were adapted to each other, and that the young animal's digestive tract and mother's milk complemented each other; also, that until development of the animal was complete it should be looked upon as being not separate from the mother, and that the ordinary laws of dietetics did not apply to it. When it ceased to be dependent on the mother and had a complete digestive apparatus, these laws would apply, as we find they actually do.

In infant feeding, therefore, we should look upon the infant as having three stages of development and nutrition. (1) A pre-placental stage of about three months. (2) A placental stage of about six months. (3) A mammary stage of about ten to twelve months.

When a baby is born much before term it is a difficult matter to save it, as it is not ready for the mammary stage. A baby that is early deprived of the breast should be looked upon, from a nutritional standpoint, as premature, and not at all fitted to be separated from its mother, as the digestive tract is not fully developed.

Colostrum is adapted for absorption and requires little or no digestive effort. It is first secreted about the third or fourth month of pregnancy. When the infant is born, its digestive apparatus is so far completed that it requires colostrum for but a few days, when normal milk is secreted. Here is a plain case of the mother adapting herself to the infant, which shows that, while at the breast, the infant must be looked upon as still in a way attached to the mother. The character of the milk does not now change to any great extent but the infant develops until its digestive tract is completed, when the milk supply normally fails and the infant commences to eat the same food as the mother.

Now, if instead of directing our entire attention to the chemcial composition of the milk, we study the changes the milk undergoes under the action of the baby's digestive juices, we will find that milk has a function aside from that of nutrition, and this function is principally connected with the proteids. While the stom-

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ach of an infant is formed at birth, its function is not developed Wiedersheim[4] states that a stomach that does not secrete the normal digestive juices should be looked upon as a widened section of the enteric canal, and from this standpoint some animals have no stomach. Strictly speaking, then, an infant has no stomach at birth, as it does not secrete pepsin and hydrochloric acid, but a dilated sac that develops into a true stomach during the suckling period.

It is impossible to make experiments with human milk and the digestive juices of infants, but it is possible with cow's milk and a calf's stomach.

During the time the calf is receiving colostrum from the cow it has little gastric secretion, but as the mammary secretion changes into milk, the secretion of rennet commences in the calf's stomach and is plentiful when the milk flow is established in the cow. We find the action of rennet derived from a calf's stomach is to convert the casein of cow's milk into a solid which occupies the same volume as the milk did. Later on the secretion of pepsin and hydrochloric acid takes place in the stomach.

During the past year, Van Slyke and Hart[5] have shown that after paracasein or curd has been formed by the action of the rennet on the milk, the curd combines with acids to form definite salts as mono or dichlorid, or lactate of paracasein, which is the first step in the process of gastric digestion, the pepsin then attacking the combination of the curd and acid; but, before the acid has combined with the curd, the pepsin does not act, digestion being entirely intestinal. This has also been observed by Babcock, Russell, Vivian and Hastings.

The tough, semi-fibrous curds of cow's milk are a combination of paracasein or rennet curd, and acid. Woman's milk, with rennet and acid, curds in loose flakes and I have never seen it act in any other way, even when strong rennet solution was used, with dilute hydrochloric acid, to faint acidity to litmus.

During the time an infant or calf is normally suckled there is developed a true stomach; that is, glands that secrete pepsin and hydrochloric acid are developed. Then the milk supply naturally fails and teeth and salivary glands develop.

During the process of mammary development we find that at first the young animal receives readily absorbable food, colostrum; as the mammary secretion changes into milk the motility of the stomach is developed by the milk changing into a soft solid

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which can easily pass into the intestine and which is the prototype of chyme. As the pepsin and hydrochloric acid begin to be secreted, the milk curds are toughened by the acid, and the pepsin then attacks them. By the time the teeth appear, the functions of the stomach are so well developed that the young animal can eat and digest with its mother. The milk of each type of animal curds in such a way that it will develop the motor and digestive functions peculiar to that type of animal, and it is for this purpose that the proteids, which produce the curds, differ. From these facts, which I think are well established, it seems to me some valuable principles can be deduced, which will tend to simplify the teaching of infant feeding.

(1) An infant undergoes three stages of development and nutrition: (*a*) Pre-placental. (*b*) Placental. (*c*) Mammary: and should be looked upon as attached to the mother in all three.

(2) At the beginning of the mammary stage, an infant has only the rudiment of a true stomach.

(3) During the period of mammary development, the mother first changes the character of the infant's nourishment from colostrum to milk, and then the infant's digestive secretions so change the character of the milk, that, as the digestive juices increase in quantity and strength, the work of digestion is not thereby lessened, but rather increased, as the stronger the gastric juice becomes, the tougher the milk curds become, owing to the acid combining with the curds.

(4) The milks of all animals will produce good tissue. They differ in composition according to the rate of growth of the young animals. Their proteids differ in accordance with the type of digestive tract they are to develop.

(5) No known method of procedure will convert cow's milk into human milk. All methods of artificial feeding must have in view the production of food that will adapt itself to the developing digestive tract. All substances aside from breast milk are foreign to the infant's digestive tract, and may at times cause disturbance.

(6) In artificial infant feeding, certain minimum quantities of digestible proteid, fat, carbohydrates and mineral matter are needed. The mean composition of breast milk will serve as a general guide to the nutritive requirements of infants.

(7) Milk must be the basis of an infant's food, not alone because it contains animal proteid, but because it contains the only

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available form of proteid that possesses the function of developing the digestive tract.

(8) The proteid of cow's milk, which was intended to develop a calf's stomach to digest grass, must be modified or adapted to the infant's stomach.

(9) This may be done by chemically or mechanically altering the character of the curds, by diluting milk with either alkalis or gruels.

(10) When, for any reason, a sufficient quantity of proteid of cow's milk cannot be given, it must be supplemented by other forms of nucleo-albumins until the normal quantity can be digested. It is a gross error to feed too low proteids of milk simply to overcome indigestion.

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DISCUSSION.

DR. ROTCH.—I think Dr. Chapin should be congratulated on this very laborious work and the way in which he has treated the subject.

The infant may not have the stomach of an adult but it has a stomach when it is born and the milk should be made to approach as nearly as possible in its proteids to what we get in human milk, where we have a large proportion of the whey proteids and a small amount of caseinogen. I have found it a great advantage to use what are called the split proteids, namely, various combinations of the whey proteids and of caseinogen. As to the colostrum, the idea has been growing more and more that it is an abnormal condition and that it has not been put there for any special purpose.

DR. KOPLIK.—I think the point made by Dr. Chapin is very well taken, although it has been made many times before. Hammarsten has pointed out that the casein of cow's milk is certainly not the same substance as casein of woman's milk. Although I am a great admirer of the whey method of infant feeding, and

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wish it could be made more practical, yet after you have your whey and your proteids, you have added caseinogen of cow's milk in order to bring the amount of caseinogen to equal that of human milk. In other words you are working with caseinogen foreign to the infant's stomach.

DR. ROTCH.—Of course, we use good woman's milk when we can get it; but when we cannot get it, I do not see why we should not use the next best substitute simply because it differs in a certain degree from human milk. I personally have found the various combinations of the whey proteids and caseinogen very useful and satisfactory. We know that the caseinogen is not the same in cow's and human milk, but we should use it, as it is practically the nearest approach to human caseinogen which can be obtained.

DR. CHAPIN.—I am afraid that the difference in the chemistry of the proteids might form a difficulty. Recent experiments show that the nucleo albumin builds tissue and also plays an important part in developing the digestive tract, according to the baby's future diet. While whey feeding is an excellent thing for occasional use, I do not think it is safe to feed babies this way constantly; it is certainly not according to the teaching of physiological chemistry and neither is it according to clinical experience. I use it but I do not use it constantly.

My point in preparing a paper of this kind was not to say that cow's milk should not be used, but simply to point out in strong light where the difficulties lie in the proteids, and our future study must be along the line of the proteids. I wished, also, to show that we should devote a great deal more attention to the mammary gland, and try to keep our babies on the breast in every way possible. I think all of those who have worked along this line come back to that point. After all our investigations we cannot improve upon the mammary gland in the function it has to perform, not only in giving the food principles in right proportion, but in developing the digestive tract of the infant for its future diet.

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CONCERNING THE ACCURACY OF PERCENTAGE MODIFICATION OF MILK FOR INFANTS.*

BY DAVID L. EDSALL, M.D.,

AND

CHARLES A. FIFE, M.D.,

Philadelphia.

The most distinctively American principle of pediatric practice is the accurate modification of milk for infants by percentage formulæ. The scientific propriety of this method needs no demonstration, and it is used in this country by almost all careful practitioners who have any skill in the feeding of infants.

In spite of the general use of percentage feeding, however, there is no record of any comprehensive study of the actual composition of the milk mixtures made by this method; and, except for Wentworth's analyses, which relate solely to laboratory modifications, there has been no extensive attempt to determine whether the mixtures actually correspond closely with the prescribed formulæ. That is, it has been customary to order a certain formula, and to proceed as if the infant were getting that formula, the accuracy and conscientiousness of the person making the modification being usually taken for granted; while the fat and proteid content of the milk and cream are considered to correspond to certain accepted average figures. In other words, the method is known to be theoretically reasonably accurate, the practical results have usually been taken on faith. A conspicuous exception to this laxity of method is found, in Philadelphia, in those that use "certified" milks; for the chemical constitution, as well as the bacterial content of these milks, is kept constantly under control by a commission of disinterested physicians. A further important method of avoiding inaccuracies is in the use of laboratory modifications. Certified milks are, however, not available in many cities, and are never available in the smaller towns or in the country, except when a city that has a supply of such milks is not far distant and the family can afford the very considerable expense of having certified milk shipped to them—a course that is impracticable in most instances. Laboratory modifications, also, are used only by a somewhat limited number of physicians. Hence, in the majority of instances, the statement that the composition of the milks and creams used is unknown, is correct; for few practitioners determine for themselves even the percentage of fats.

* From the William Pepper Laboratory of Clinical Medicine. Phoebe A. Hearst Foundation.

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We have undertaken a series of chemical examinations of milk mixtures, with the purpose of learning whether there are any important errors in the percentage method of feeding, as it is usually carried out; and if errors exist, to determine their sources and, if possible, the manner in which they may be corrected. Our purpose was to get some information concerning the results of several of the most important methods of percentage modification, and to determine which are the best and which are the least reliable. In carrying out this purpose we have analyzed 88 different milk mixtures.

Wentworth's figures relate solely to laboratory modifications, and throw no light upon the accuracy of home modifications. He gives a few analyses of modifications made by himself; but, since they were made up from milks and creams that he analyzed himself, the correctness of these modifications depended solely upon the care that he used in measuring and mixing, and they do not indicate the conditions that one would find in practice with milks and creams of more or less doubtful composition, and with modifiers that may vary from extreme intelligence and devotion to utter stupidity and lack of conscience.

The two important sources of error that at once occur to one have already been indicated. First and foremost is, of course, variation in the composition of different milks and creams; particularly in the fat content of the creams. We have, for instance, found ordinary market creams, sold as "20 per cent. cream," to be as low in fat as 13 per cent. and as high as 26.5 per cent.—a variation of over 100 per cent. from the lowest point to the highest; and this, of course, entails a similar variation in modifications made from these creams. We have also known the fat content of milk to range from 2.5 per cent. to 8.8 per cent. in ordinary market milk—a matter of equally serious importance when whole milk or top milk is used in modifications. The proteid is subject to far less marked variations in unadulterated milk, but variations do occur. We have seen a range, within a brief period, from 2.85 per cent. to over 4 per cent. in mixed milk from one dairy of extremely good general quality; and the effect of such a variation, or of even a somewhat less pronounced one, upon a modification is at once evident. The proteid of cream is sometimes said to be less variable; but this is not an accurate statement, for marked variations occur, and especially with decided variations in fat percentage, there are usually coincident contrary variations in the proteid.

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The other chief source of error in modifying milks is undoubtedly to be found in the person who does the measuring and mixing. That carelessness may often be discovered, if looked for, every one will admit. That confusion and lack of understanding, even in persons of the more intelligent classes, is an equal source of error, will, we believe, be shown by our figures.

In order to meet these chief opportunities for error and to determine their effect, we have analyzed milk-mixtures made by supposedly capable persons and by others of unknown reliability; and also mixtures made, on the one hand, from milks and creams known to be of reliable chemical composition and, on the other, of ordinary market milks and creams. We are indebted to Dr. Griffith, Dr. Hamill, Dr. Westcott, Dr. Hand, Dr. Ostheimer and Dr. Davisson for furnishing us with a number of the samples of mixtures from hospital and private patients.

In a general way, our analyses fall into the following four series:—

(1) A series of home modifications made from milks and creams of known composition.

(2) A series of home modifications made from ordinary market milks of unknown composition.

(3) A small series of modifications made from Dr. Ladd's card, chiefly using top milk and the lower fat free milk.

(4) A series of laboratory modifications.

In the first and second series, the modifications were made by persons of varying capability. In one or two instances, the infants' fathers, who are physicians, modified the milk themselves. Usually it was done by a trained nurse or by the mother, the intelligence of the latter varying, in the different instances, from a high degree down to that of a stupid dispensary patient. The third series we modified ourselves, chiefly to test the accuracy of modifications made from top milk and the underlying, supposedly fat free, milk.

In all cases we determined only the fat and the proteid. The sugar is of less consequence in digestive disturbances, and, furthermore, the amount is more directly under our control, since we do not use a natural product containing a varying percentage of sugar, as we do with the fat and the proteid, but chiefly dry milk sugar. The fats and the proteid are the important constituents, and these we estimated accurately.

The method used for the proteids was determination of the nitrogen by the Kjeldahl-Argutinsky method, the value obtained

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being multiplied by the factor 6.38. This gave the proteid figures. We always used the mean of two control estimations. The reading of the titration in the control estimations rarely varied 0.1 cc. ($^4/_{10}$ normal solution).

The fats were estimated chiefly by the Leffmann-Beam method, frequently controlled by ether extraction. In a few instances, the latter method was used alone. It is important to note that in using the Leffmann-Beam method we, in the first place, always warmed the mixture to be analyzed to 15°C., and used a carefully calibrated pipette in measuring. We always used exactly 9 cc. of concentrated sulphuric acid of constant strength; and the dilute sulphuric acid used was always of constant strength. Two control estimations were frequently made, and always corresponded within 0.05 per cent.; and the result can practically not be read more accurately than this. If a new stock of concentrated or dilute sulphuric acid or of the hydrochloric acid amyl-alcohol mixture became necessary, the accuracy of this new stock was always tested by making control ether extractions. The ether extractions were always made in the Soxhlet apparatus after drying on asbestos (Babcock method).

In the following tables, we have introduced on the left the percentages of fat and proteid supposedly contained in the milk mixtures; on the right are those actually determined to be present. The letters L-B indicate the Leffmann-Beam process; eth. ext. indicates ether extraction.

SERIES I. contains mixtures made from certified milks:—

SERIES I.—CERTIFIED MILKS.

	Calculated.		Determined.		
			Fat.		
No.	Fat.	Proteid.	L. & B.	Eth. Ext.	Proteid.
1	3	1.50	3.2		Sample too small for estimation.
2	3.8	2.16	3.0	2.965	2.2151
3	4.16	1.50	4.4		1.3396
4	6.6	1.59	5.3		1.57
5	6.6	1.59	5.0		1.542
6	3.0	1.50	2.82		1.6335
7	3.0	1.50	2.80		1.555
8	3.1	1.60	2.85		1.429
9	3.1	1.60	2.80		1.597

SERIES II. were modifications of ordinary market milks and creams. The first ten were modified at the patients' houses, in

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all cases by the mothers. The remainder, the larger number, were modified in children's wards in hospitals by nurses especially trained in the care of children.

SERIES II.—MARKET MILKS MODIFIED AT PATIENT'S HOUSES.

No.	Calculated. Fat.	Calculated. Proteid.	Determined. Fat. L. & B.	Determined. Proteid.
10	3.0	1.0	3.4	0.7146
11	4.0	2.0	5.6	1.375
12	4.0	1.0	4.3	0.857
13	2.7	1.8	2.9	1.75
14	2.5	1.0	2.3	1.071
15	4.0	2.0	4.5	1.804
16	4.0	2.5	5.4	2.006
17	3.5	2.5	3.95	2.009
18	3.0	1.0	2.8	1.036
19	2.0	0.75	2.35	0.7146

MARKET MILKS MODIFIED IN HOSPITALS BY TRAINED NURSES.

No.	Calculated. Date.	Calculated. Fat.	Calculated. Proteid.	Estimated. Fat. L. & B.	Estimated. Fat. Eth. Ext.	Estimated. Proteid.
20	1.16	4	2	3.6		1.822
21	1.16	3.56	1.25	2.4		1.750
22	1.17	3	1	2.4		.7503
23	1.17	4	2.25	2.9		1.786
24	1.20	4	2	2.4	2.28	1.6792
25	1.20	3	1.25	2.4		1.2147
26	1.20	4	1.25	2.7		1.6335
27	1.20	3	1		2.802	.9646
28	1.20	3	1.25		1.806	1.036
29	2.2	4	2.5		2.72	2.1794
30	2.16	4	2	3.2	3.18	1.8935
31	2.16	3	1	2.2	2.24	1.036
32	2.19	4	2	3.2	3.4	
33	2.19	2	0.5	1.30	1.28	
		3.5	1.25	1.9		1.435
34	3.12	3	1	4.3		.786
35	3.12	3.5	1.5	4.5	4.42	1.2862
36	3.12	2.5	1.5	3.1		1.1432
37	3.17	2.5	1.5	3.2		1.2861
38	3.19	3.5	1.5	4.2		1.1432
39	3.19	3	1.5	3.8		1.1432
40	3.19	4	1	3.8	3.75	.8574
41	3.20	2	3	6		2.286

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SERIES III.—Chiefly modifications made by ourselves from Dr. Ladd's card. Nos. 52 to 55 inclusive were made by using Baner's formula.

SERIES III.—MODIFIED BY THE AUTHORS.

No.		Calculated.		Determined.	
		Fat.	Proteid.	Fat.	Proteid.
42	Certified milk, "B" $^{4}/_{12}$, 1903, presumably 10 per cent. cream, twelve hours standing. Determined fat percentage of cream, 7.6	2	1	1.2	.912
43	Same milk, $^{4}/_{12}$	2	1	1.6	.928
44	Same milk, $^{4}/_{14}$, 1903, presumably 10 per cent. cream. Milk stood ten hours. Fat percentage 11.1	3	2	3.25	1.929
45	Certified milk "A," presumably 10 per cent. cream. Milk stood twelve hours. $^{4}/_{12}$, 1903	2	1	1.8	.9746
46	Same, $^{4}/_{14}$, 1903	3	2	3	1.8578
47	" $^{4}/_{15}$, 1903	4	2	3.9	
48	" $^{4}/_{16}$, 1903	3	2	3	1.786
49	" $^{4}/_{17}$, 1903	2	1	1.85	1.1003
50	" $^{4}/_{18}$, 1903		2	3.15	1.9649
51	Milk of unknown fat percentage stood eleven hours, giving supposedly 10 per cent. cream	3	2	2.65	1.826
52	Certified (fat-free) milk and	2	1	1.8	1.0003
53	16 per cent. certified cream.	3	2	3.1	2.036
54	Baner's formula	4	2	3.8	1.929
55	Unknown market milk and supposed 16 per cent. cream. Baner formula	3.5	1.25	1.9	1.435

SERIES IV.—Laboratory mixtures ordered for this purpose to be sent to a city address. A number of specimens of each modification were examined, since this would lead to more proper conclusions, whether favorable or unfavorable, than would the ex-

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amination of single specimens. The percentages were run up rapidly, for the purpose of testing the results.

SERIES IV.—LABORATORY MODIFICATIONS.

No.	Calculated.		Determined.		
	Fat.	Proteid.	Fat.		Proteid.
			L. & B.	Eth. Ext.	
56	1.5	.75	1.3		.822
57	1.5	.75	1.3		.8217
58	1.5	.75	1.4		.8217
59	1.5	.75	1.2	1.3	.7503
60	1.5	.75	1.3	1.3	.7146
61	1.5	.75	1.1		.8574
62	1.5	.75	1.45		.8217
63	1.5	.75	1.30		
64	1.5	.75	1.50		.8574
65	1.5	.75	1.50		.9288
66	2.	1.25	2.2		1.2147
67	2.	1.25	1.9		1.2504
68	2.	1.25	1.9		1.2147
69	2.	1.25	2.4		1.2861
70	2.	1.25	1.6		1.036
71	2.	1.25	1.7		1.2147
72	2.	1.25	1.9		1.0717
73	2.5	1.50	2.4		1.2504
74	2.5	1.50	2.6		1.3891
75	2.5	1.5	2.6		1.2861
76	2.5	1.5	2.6		1.4098
77	2.5	1.50	2.2		1.143
78	2.5	1.50	2.6		1.3
79	3.5	1.75	3.55		1.6335
80	3.5	1.75	3.3		1.6335
81	3.5	1.75	3.3		1.544
82	3.5	1.75	3.25		2.2151
83	4.5	1.75	3.9		2.1794
84	4.5	1.75	4.2		1.544
85	4.5	1.75	3.5		1.536
86	4.5	1.75	3.8		1.7049
87	4.5	1.75	3.9		1.557
88	4.5	1.75	3.7		1.562

In interpreting these results, one must first realize clearly that *absolute* accuracy can never be demanded or expected in the

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modification of milk. Consequently, if figures obtained by the chemical examination of modified milks occasionally vary a few tenths of 1 per cent. from the calculated figures, and only a few tenths, one is not justified in condemning the mixture in any way; on the contrary, if these variations are only occasional, the mixtures should be considered to be excellently made. This statement is based upon the well-known fact that the composition of milk and cream varies more or less widely, even when the product of one excellent dairy is used. Therefore, modifications made from milk and cream will vary somewhat, no matter how great the care used in preparing them. It is common knowledge that the fat of whole milk, even when from a constant source, may vary widely within a short time, and even from day to day. To quote our own results, we have, among others, several series of figures from estimations of the milk from one excellent dairy, made for days or weeks at a time, which shows variations as marked as from 3 per cent. to 5.5 per cent. of fat within a very few days; and these figures are not at all remarkable. The Milk Commission of the Philadelphia Pediatric Society allows the fat of the milks that it passes as "certified" to vary between 3.5 per cent. and 4.5 per cent.; and yet, although these milks are produced under conditions that are as nearly ideal as it is practicable to make them, the fats not infrequently pass these limits slightly and require reexamination. A very appreciable error would result from these variations in mixtures made from whole milk; and when fat free milk is used and the fat is obtained entirely from cream, an error of as much as 0.4 per cent. is possible in modifications with fairly high fat percentages. The fats of the Philadelphia certified creams are allowed to vary between 14 per cent. and 18 per cent. and do occasionally vary to this extent, even though they also are produced under most excellent regulations.

As to the proteid, it is often said that this varies but little—and this is true, as compared with the fat; but it varies enough to make an error of several tenths of 1 per cent. possible in modifications of moderate strength. The Philadelphia milk commission allows a variation from 3.5 per cent. to 4.5 per cent. in proteid, as well as in fat; and no narrower limit could be maintained. As to excellent market milks, we have figures for several hundred estimations, made by us, that show even wider variations. Among these figures are many that were made day after day in metabolism experiments; and these show variations even as great as 1.1 per

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cent., on two successive days, with thoroughly mixed milk from the same dairy.

It is evident, then, that in the modifications themselves, if they are of moderate strength, an extreme variation of as much as 0.3 per cent. in the proteid and certainly quite as large a variation in the fat, may be due purely to uncontrollable variations in the milk and cream; and such variations are not subject to criticism. Again, it may be properly said that a reasonably conscientious and careful modifier may often make an error of as much as one dram in measuring, and this would sometimes, in twenty-ounce mixtures, increase the fat error as much as 0.1 per cent. If a similar error were made in both milk and cream, it would add at least 0.05 per cent. to the total proteid error; and this would make the possible excusable error with the proteid as much as 0.35 per cent.

We wish it to be clearly understood that the statement that has just been made is not intended to indicate that wide variations of fat and proteid are constantly occurring, even in carefully prepared modifications. Such a wide range is permissible only in mixtures ranging from a moderate strength up to a high formula, and not in very low formulæ; and it is never permissible for them to occur very frequently. The latter would mean that poor dairy products were being used or that the modifier had been careless. We do mean, however, that in formulæ with fats or proteids running as low as 1.25 per cent. there may be an occasional entirely excusable error of fully 0.3 per cent. in either fat or proteid. In lower modifications, which are the most important from this standpoint, the excusable error would decrease in accordance with the greater dilution; in higher modifications. a somewhat larger error may be excusable. Errors as marked as this should occur only occasionally, because extreme variations in good milk are only occasional. The fats, however, show a wide range more frequently than do the proteids; consequently, moderate errors in the fats may be expected with some frequency with mixtures containing high or moderately high fats. The fats of milk or cream may go either up or down; consequently, the error in the modification may do the same. The proteids not only do not vary so frequently, but when they do vary they will usually be below what they are calculated to be, because the figure generally used in computing the formula for proteid is 4 per cent., and the actual proteid is usually somewhat below that point and is much more likely to go still lower than to go above 4 per cent.

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Considered from this standpoint, our figures show the following facts:—

The first series of home-modifications, made under favorable circumstances—*i.e.*, from certified milks and creams and by persons of intelligence—show satisfactory accuracy, as far as the proteid is concerned, the widest variations above or below the prescribed formula being slightly within 0.2 per cent. The fats vary widely, there being errors of 0.8 per cent., 1.3 per cent., and 1.6 per cent. in this small series. These errors were almost entirely due to the modifiers who were in these cases usually intelligent mothers. We analyzed the creams used in the two worst instances and found them to be almost exactly of the percentage that they were supposed to be.

In the second series, the home modifications made from market milks and creams, the results are extremely erratic. The proteids vary from the prescribed formulæ as much as 0.8 per cent. downward, and 0.4 per cent. upward; while it is rare for the fats to come within 0.5 per cent. of the amount prescribed, and in a large proportion of instances the error is so large that the actual result scarcely shows any resemblance to the formula aimed at. The error in the fats is certainly due, in large part, to the cream used; and to some extent, to the milk. We analyzed the milks and creams used in the hospital modifications. The figures for the fat of the cream used in Nos. 20 to 33 of this series show 13.75 per cent. instead of the supposed 20 per cent.; and all these figures for fat in the modifications are too low. On the other hand, the fat of the cream used in Nos. 34 to 41 ran at 21.5 per cent. and 22.2 per cent., instead of 16 per cent.; and the fat of the milk, at 6.2 per cent. and 8.8[1] per cent., instead of 4 per cent. In all these modifications, the fat is too high. The results with the proteid are also partly due to the fact that the proteid of the milk and cream was running low; this is particularly true of the second set, in which the milk proteid was at one estimation 2.41 per cent.

The results cannot, however, be attributed entirely to variations in the milk and cream. The dates on which these modifica-

1 It is probable that this "milk" was, in part at least, old cream watered, with perhaps some separated milk added. This is indicated by the very high fat, the low proteid (2.4 per cent.), and the fact that a bacterial count showed enormous numbers of bacteria. It came from a supposedly reliable source, but the milk supplied the general public from the same source contained on several examinations about 3.6 per cent. of proteid and 4 per cent. of fat.

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tions were made have been given, in order to demonstrate that the modifier was at fault. They show this clearly. On individual days, when exactly the same milk and cream was used in the several modifications made on that day, it may be seen that the different mixtures showed marked errors of very different kinds. On January 20th, for instance, a supposedly 4 per cent. fat-mixture contained 2.7 per cent.; while a supposedly 3 per cent. mixture at the same time contained 2.8 per cent., and another 3 per cent. mixture contained 1.8 per cent. On March 19th, a supposedly 4 per cent. and a supposedly 3 per cent. mixture contained an equal amount of fat—3.8 per cent. There are several similar examples of the same thing. On March 20th, indeed, a mixture of odd proportions (2.6.3), which was ordered purely for testing the accuracy of the modification showed 6 per cent. fat, instead of 2 per cent. As to the proteids, on January 16th, a supposedly 1.25 per cent. mixture contained 1.75 per cent.; on January 20th, a supposedly 2 per cent. proteid showed 1.67 per cent.; on the same date, a proteid calculated at 1.25 per cent. contained 1.63 per cent., another calculated at 1.25 per cent. was found at 1.03 per cent. The proteid of the milk used in the last mentioned modifications we have repeatedly estimated and found to be regularly between 3.4 per cent. and 4 per cent.

The variations in the mixtures were undoubtedly due to the modifier, and were the result of either carelessness or confusion. Carelessness probably played a part, but we believe that a lack of clearheadedness had more to do with the results. The figures that we have referred to in detail are all from modifications made by nurses especially trained in the care of infants and children. We know the nurses that did this work, and know them to be reasonably conscientious and trustworthy; and yet the results are attributable to them. They can scarcely be considered to be due to anything but lack of clear understanding and to confusion in measuring off the various constituents of the mixtures. This is a matter of importance that will be briefly referred to later.

As to the third series, the time at our disposal prevented our examining many specimens; and we have, therefore, little right to speak concerning the accuracy of mixtures made from top milk. Dr. Ladd's extremely convenient card, with its formulæ ready made, is, in itself, evidently entirely accurate. When the top milk contained about the amount of fat that it was supposed to contain (Nos. 44 to 50), the mixtures corresponded closely

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with the calculated formulæ. With the first two mixtures made from certified milk "B," however (Nos. 42 and 43), the results for fat (1.2 per cent. and 1.6 per cent., instead of 2 per cent.) show decided error; especially the first.

These results, few as they are, show that with one milk, such as certified "A," the results by the top milk method may be excellent over a series of days; while with other milk of fine quality the result may be decidedly erratic. The results that have been obtained by different investigators of the fat content of the top milk are somewhat confusing. Several authors agree that the fat content is reasonably constant, but give somewhat different figures for the same quantity of top milk. Others have no faith in the reliability of the method. Our own small series of figures and—much more than this—the knowledge gained from dairy-chemists of wide experience lead us to believe that the use of top milk is unsatisfactory, unless the fat content of the individual top milks used is controlled to some extent by fat estimations. Milks, in their natural state, certainly differ to some extent in the rapidity and completeness with which the cream rises. It seems probable, though it has not yet been definitely proved, that in most fresh milks this is sufficiently constant to give wholly satisfactory modifications. Other milks, in their natural state, probably yield their cream more slowly or more rapidly than the standard; and probably with varying rapidity at different times. At any rate, it is well known to dairy chemists that heating milk causes the cream to rise very slowly; and it is likewise well known that some milk dealers pasteurize their milk, either covertly or openly, and this is nowadays often done at temperatures that cause no change in the taste of the milk, and it is therefore often not known that it has been done. Hence, this source of error in using top milk may be met with at any time in market milk, and is an important argument against the method. On the whole, we believe that it is a method of doubtful value, as our knowledge stands at present. It has, at least, been shown that it is not always reliable; and this means that if it is to be used with any confidence, the fat of the individual top milk in use should certainly be estimated once, and better, several times, in order to see how it is running.

We agree with many others that the most accurate method is to use a cream of known fat percentage from an entirely reliable source. The objection has repeatedly been raised that creams sold

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as containing a certain percentage of fats have been found, on analysis, to vary widely from this percentage. This is quite true of many market creams; it is, however, not true of creams from all sources. The Philadelphia milk commission now certifies to several creams, and this means that analysis of these creams constantly shows that their fat-content runs within narrow limits. Equally reliable cream may be obtained in almost any of our large cities and in many smaller towns.

It is also asserted by some writers that in the process of separation the emulsion of the fat is altered, and that a milk modification made from it is less digestible than one made from a gravity cream. There is no good proof of the truth of this statement. If, however, it impresses one as being important, there is in Philadelphia a reliable (certified) gravity cream of constant 16 per cent. fat content; and such creams may be obtained elsewhere.

As to the fourth series, the laboratory modifications, the proteid variations in this series are slight, and are worthy of remark in only three instances: *i.e.*, Nos. 77, 82, and 83. The variation of 0.36 per cent. in No. 77, in a proteid formula of 1.50 per cent., certainly falls within the limit of occasional unavoidable error. The errors of 0.46 per cent. and 0.42 per cent., with a proteid formula of 1.75 per cent., are perhaps a little beyond the range that is permissible; but they are only slightly so. That these errors may be attributed largely to unavoidable variations in the milk and cream is made probable by the constant way in which the other figures run.

The figures for the fat are a little less satisfactory; but all the figures are within the range of unavoidable error, except those for the 4.5 per cent. fats—those numbered 83 to 88. In this set, the fat regularly ran very low; except in No. 84, when it was 4.2 per cent. Some estimations that we made of the fat of the cream from the laboratory company at about the same time showed that it was running about 2 per cent. below its usual figure. This would explain the major portion of the fat error in the modifications, leaving the smaller portion still unexplained. On the whole, the results are excellent, particularly with the low formulæ, which are the more important. It is but fair to say, also, that by subsequent inquiry we learned that the regular modifier had been ill for a portion of the time during which we were making these estimations; and the results would perhaps have been even more

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accurate, had he not necessarily been replaced by a substitute, who, while trained in the work, was less accustomed to it.

These analyses of laboratory modifications are much more favorable than Wentworth's. The difference in our results may, perhaps, be due to the fact that the modifications examined by us were better prepared than those that Wentworth analyzed; but a more definite and satisfactory explanation may, we think, be found in the differences in the methods used. As already stated, we used various precautions in our Leffmann-Beam fat determinations, and frequently controlled them with ether extractions. Wentworth, who used the Babcock method, which needs similar precautions, makes no statement of having adopted these precautions; and particularly, did not use control ether extractions. The proteid we determined directly by Kjeldahl nitrogen estimations. Wentworth used the method of difference, which includes in bulk all errors made in the fat, total solid, ash, and sugar determinations; and the error in sugar determinations, in particular, is likely to be considerable when the polariscope is used. We cannot emphasize this last statement more strongly than by referring to Stohmann, who, in his excellent article in Musspratt's *Encyclopädie der Technischen Chemie,* states that he cannot recommend polariscopy at all for determining the sugar in milk, as the error associated with it is likely to be even great enough to cover the whole amount of sugar in the milk. This very decided view is not shared by all dairy chemists, and the error in the special polariscopic method used by Wentworth is undoubtedly much less, but even that method is known[2] to be subject to considerable error, and polariscopy is always exceedingly untrustworthy unless one has extensive and continued experience in its use. The effect of a much less marked error in polariscopy than that attributed to it by Stohmann is evident and striking, if the method of difference is used in estimating a proteid of, for example, 1 per cent. Indeed, we are not aware that the difference method is considered reliable by any authorities.

The general conclusions that we believe our work justifies are: —(1) Milk modifications, under the best of conditions, will often vary 0.2 per cent., occasionally 0.3 per cent. from the prescribed formulæ, with modifications of medium strength. With low modifications, the error will be correspondingly less; and with high modifications, correspondingly somewhat greater.

2 Richmond, *Dairy Chemistry.*

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(2) Home modifications are within the limits just mentioned, trustworthy, if they are made by a clear-headed and careful person, from milks and creams of a chemical composition that is known to be reliable.

(3) If the composition of the milks and creams used is not known, or if one cannot be sure that the modifier is careful and clear-headed, home modifications are likely to vary greatly from the formulæ prescribed.

(4) Laboratory modifications appear from our figures to be satisfactorily accurate; and unless the milks and creams, as well as the person doing the modifying at home, can be fully trusted, laboratory modifications, when available, are much more reliable than home modifications.

Undoubtedly, the most satisfactory method of having milk modifications prepared is to have a thoroughly capable person make them at home, from milk and cream of reliable composition. In this case, the whole process can be kept under direct supervision. This means, however, that milks and creams that are constantly watched, as are the certified milks and creams in Philadelphia, must be used; and that a clear-headed mother or nurse must do the modifying. To trust the modifying to a readily confused mother, trained nurse, or nursery governess, or to most nursemaids, would make the results very doubtful. We confess that our analyses while not lessening our faith in the devotion of mothers and nurses, do make us somewhat doubtful of the accuracy of many of them in making milk mixtures. The analyses of their modifications demonstrate how important it is, in having home modifications made, to have all the small details and, above all, the amounts of the different ingredients, carefully written out, or filled out on printed blanks, in order to guard, as far as possibe, against confusion. Nothing should be left to the modifier's judgment or memory.

The figures that we have given demonstrate, also, that it is careless practice to order a certain formula for an infant, and to take it for granted that this formula is actually being given to the child. It is, of course, difficult for those in general practice if they have not even the small facilities necessary in doing such work, to determine the fat content of the milk mixtures that are actually being given to the infants under their charge. It would, however, be very easy for many such physicians to have this done by an assistant. Those that are especially interested in pediatric

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practice can hardly show proper interest in their work, unless they often do this or have it done. Simple centrifugal estimations of the fat are certainly quite sufficient for clinical purposes, as an indication of the accuracy of the mixtures; though an occasional proteid estimation, if one has facilities for it, will add to the effect. The knowledge that this is likely to be done at any time will necessarily have an excellent moral effect upon any modifier who has a tendency to carelessness; and the somewhat general use of such controls would go far toward eliminating any question as to the accuracy of percentage modifications, whether made at home or in a laboratory. At the same time, it would give one constant knowledge as to their accuracy in any individual case. The apparatus required is cheaper than that used for ordinary examinations of the blood; the method requires less time, and it gives good results. It is already occasionally used by some physicians, but is not often employed by any considerable number of them.

Finally, it is proper to insist again upon the fact that we have already repeatedly mentioned, *i.e.*, that there is a very great advantage, from the chemical standpoint, as well as from the hygienic, in the use of milks and creams that are under constant professional control and are certified to by members of the profession as being reliable. Such milks are already available in a number of our cities; those interested in pure and reliable milk in some other cities are preparing to institute their use in those cities; and it may well be urged upon the members of the profession elsewhere that they should offer the same protection to their patients. These certified milks and creams have been received with a curious degree of skepticism by many physicians, but they stand the tests put upon them admirably. There are on sale in Philadelphia six certified milks, and three certified creams. We have made a large series of analyses of these products, and know that, except for occasional slight and inconsequential variations, they do not pass the narrow limits imposed upon them. They are, therefore, suited, chemically, for milk mixtures, as are few others in this city; and as no others are known by the profession to be. The records of the veterinarian and of the bacteriologist of the Philadelphia milk commission testify as to the care given these milks in their production and sale and demonstrate their remarkable purity from the hygienic standpoint. The best indication of their condition as they reach the consumer is the number of bacteria that they then contain. The limit placed upon them is 10,000 bacteria to the

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cubic centimetre. They are not allowed to run above this limit; and are, indeed, usually much below it. Several of them regularly and consistently, at all the examinations made, have shown only a small fraction of that number. It should be remembered that most market milks show hundreds of thousands of bacteria to the cubic centimetre, and that, as delivered in large cities, many of them show millions. One of the latest additions to our knowledge concerning this matter is Wall's statement that Gehrmann recently found the average in fifty specimens of Chicago market milk to be over 2,600,000; and only three were below 100,000. Similar figures have been obtained in other large cities. Such figures demonstrate the wretched and dangerous hygienic conditions that attend the production and sale of most market milks; and, at the same time, they show that the strictest care must be used with milks that are not passed if they contain more than 10,000 bacteria to the cubic centimetre. Certified milks are not examined only once or occasionally, but at least once each month that they are sold. No one can rationally object, therefore, that these milks are not what they are intended to be, *i.e.*, of as constant a chemical composition as is possible, and—most important of all—free from dangerous contamination.

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THE DETERMINATION OF FAT AND TOTAL SOLIDS IN MILK.

BY HENRY L. K. SHAW, M.D.,
Albany, N. Y.

The amount of butter fat in milk can be determined by various methods. The principle of most of these is similar and consists of dissolving the solids, not fat, of the milk by means of various chemicals and either measuring the fat as such in a narrow, graduated tube or dissolving it with ether, gasolin, etc., and weighing it on evaporation of the solvent. The separated fat is brought into the narrow, graduated tube by centrifugal force in the Babcock, Gerber, and Leffmann-Beam tests.

In order to ascertain the relative accuracy of several of the simpler clinical tests for determining the amount of fat in milk, I made a number of comparative analyses. The following methods were employed.

THE BABCOCK TEST.—This is distinctively an American test and is widely employed by dairymen in this country. The glassware consists of test bottles, a milk pipette and an acid measure. The neck of the test bottle on which the scale is etched is three inches long and is graduated from 1 to 5 or 1 to 10 per cent., and further subdivided into tenths of 1 per cent. The tester is a centrifugal machine run by hand, steam, or motor power. The machines on the market are large and cumbersome and not at all practical for the physician's use. At my suggestion a small hand centrifuge was made by D. H. Burrell & Co., Little Falls, N. Y., which is thoroughly practical, requires the regulation size test bottles and takes up very little room in the physician's laboratory.

The test is made as follows: Seventeen and six-tenths cc. of milk are drawn into the milk pipette and run into one of the test bottles. Seventeen and a half cc. of clean sulphuric acid with a specific gravity of 1.082 are then slowly poured in the test bottle. The milk and acid are thoroughly mixed by a rotary motion,

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placed in the centrifuge and whirled for four minutes. Boiling water is then added by means of the pipette until the lower part of the column of fat comes within the scale on the neck of the test bottle. A second whirling for one minute completes the separation of the fat. The fat thus obtained should form a clear, yellowish liquid quite distinct from the acid solution. The fat is measured from the lower line of demarcation between the fat and the water to the top of the meniscus of the upper level. This test requires but one chemical and is extremely simple and easy of execution.

THE GERBER METHOD is employed a great deal in Europe and is somewhat similar to the Babcock test. The machine is large and bulky and is revolved by rapidly pulling on a strap. This is not an easy procedure and is attended with a great deal of noise. The glassware consists of test bottles with long necks graduated into fifths of 1 cc. These are stopped with rubber corks which are very apt to slip out during the whirling. There are also pipettes for the milk, acid and amyl alcohol. Ten cc. of milk are run into the test bottle and then 10 cc. of sulphuric acid are added. These are thoroughly mixed and 1 cc. of amyl alcohol introduced. The cork is inserted and the bottles reversed and placed in a cup in the centrifuge. The machine is run for four minutes and the amount of fat read from the scale.

THE LEFFMANN-BEAM TEST, or rather an adaptation, is used with the hand centrifuges commonly supplied to physicians. The scale on the neck of the test bottle is only about an inch in length and is divided so as to register 5 per cent. of butter fat in fifths of 1 per cent. The scale is so small that an accurate reading is almost impossible. The neck of the bottle being so small, the milk and reagents are introduced with some difficulty. Five cc. of milk are introduced into the tube and to this is added 1 cc. of a mixture of fusel oil, wood alcohol and hydrochloric acid. After this is thoroughly mixed sufficient sulphuric acid is added to bring the fluid in the neck of the bottle. The tube is then placed in the centrifuge and whirled for a few minutes.

THE MARCHAND LACTOBUTYROMETER is better known abroad than in this country. It consists of a glass tube closed at one end, with a narrowed segment near the open end. The narrow portion is graduated into thirty divisions. The tube is marked so that the lower portion shall contain 5 cc. of milk, the middle 5 cc. of ether, and the upper 5 cc. of alcohol. The milk is first added

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to the point indicated in the tube and a few drops of a 10 per cent. solution of sodium hydrat introduced. The ether is then added to the next division. This is thoroughly but gently mixed and when the mixture is homogeneous the alcohol is added and again carefully mixed. The ether should have a specific gravity of .725 at 60°F., and the alcohcl should be about 93 per cent. The tube is immersed in a water bath with a temperature of 100°F. for fifteen or twenty minutes and the amount of fat read off from the scale. To obtain the percentage of fat the following formula is employed in which X equals the number of divisions on the scale.

Percentage of fat = X x .233 + 1.266. This is a simple and easy method, requiring but 5 cc. of milk. Not infrequently a coagulation takes place when the alcohol is added which spoils the test and sometimes the fat fails to rise so that this test cannot always be relied upon. This is not so liable to occur with mother's milk.

THE HOLT CREAM GAUGE consists of a cylinder holding 10 cc. and graduated into tenths of 1 cc. Ten cc. of milk are poured in the bottle and allowed to stand for twenty-four hours. The amount of cream can be read off in tenths of 1 cc. The percentage of fat is reckoned on the ratio of five to three. This gives only approximate results and is not accurate enough for practical purposes. Fresh milk in which the cream has not risen is said to be necessary in order to obtain satisfactory results. It is only fair to state that the samples used in these tests were from two to five days old.

All the samples of milk examined were received from the laboratory of the New York State Department of Agriculture. They were examined by the chemist, Dr. Wheeler, and a comparison of results was not made until after the completion of the tests so as to avoid all possibility of personal bias in the calculations. The percentage of fat was determined by the state chemist according to the Adams' extraction method. This method consists in spreading the milk over absorbent paper, drying and extracting the fat with ether in a Soxhlet apparatus. The ether is removed by distillation and the fat in the flask is weighed. This procedure requires special chemical training and expensive apparatus and is not suitable for the physician's use. The results, however, are scientifically correct and were the standard in comparing the accuracy of the various tests.

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TABLE A.—COMPARATIVE FAT READINGS.

Number.	Extraction Method.	Babcock Method.	Marchand Lactobutyrometer.	Gerber Method.	Leffmann-Beam Test.	Cream Gauge.
1	3.27	3.2	2.89	2.	3.	6 (3.6)
2	3.12	3.	3.12	1.9	x	6 (3.6)
3	4.63	4.3	neg.	2.7	3.	10 (6.)
4	3.62	3.6	neg.	2.6	3.2	7 (4.2)
5	4.6	4.1	4.16	2.5	3.6	8 (4.8)
6	2.9	2.9	2.56	1.	2.	6 (3.6)
7	3.15	3.1	3.17	x	x	7 (4.2)
8	3.54	3.2	3.27	x	x	7 (4.2)
9	3.73	3.7	3.07	x	x	8 (4.8)
10	3.07	3.2	2.8	x	x	8 (4.8)
11	4.45	4.5	neg.	x	x	10 (6.)
12	3.41	3.4	neg.	x	x	x
13	4.74	4.7	4.52	x	x	x
14	4.14	4.	3.82	x	x	x
15	3.89	3.8	3.9	x	x	8 (4.8)
16	4.31	4.2	4.52	x	x	10 (6.)
17	3.48	3.4	3.124	x	x	x
18	2.7	2.6	1.95	x	x	6 (3.6)
19	3.75	4.	2.56	x	x	9 (5.4)
20	2.73	2.9	neg.	x	x	x
21	3.28	3.5	neg.	x	x	x
22	3.25	3.	2.86	x	x	x
23	3.23	3.	2.86	x	x	x
24	3.21	3.	2.86	x	x	x
25	3.87	3.8	3.37	x	x	x
26	3.94	3.8	3.6	x	x	x
27	3.68	3.6	3.59	x	x	x
28	3.53	3.4	neg.	x	x	x
29	2.54	2.4	neg.	x	x	x
30	3.93	4.	neg.	x	x	x

On comparing the results of these tests (see Table A) it will be seen that the Babcock test was most near the standard and rarely varied more than two-tenths of 1 per cent. The average error of the thirty tests was only seven-hundredths of 1 per cent. The Marchand test was very accurate in some instances but not nearly so uniformly exact as the Babcock test. In several instances the fat was not released, although the same sample of

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milk was tested several times. The Gerber test was not at all accurate in my hands and was discarded after six tests in this series on account of the difficulties attending its use. The object of these analyses was to show the most practical and trustworthy method of fat determination, and tests which were very inaccurate or were difficult of execution were discarded after a thorough trial. The Leffmann-Beam test was given up on account of the small size of the milk tubes.

The Babcock test, therefore, can be relied upon for accuracy and it is the simplest, easiest of operation and most economical test for the use of the physician.

The importance of estimating the amount of total solids in our feeding mixtures is not generally recognized. Some attention is paid in Germany to the caloric value of the food but very little, if any, work has been done along this line in this country. The breast-fed baby gets from 12 to 14 per cent. of total solids from the very start, while the bottle-fed baby has to get along with from 7 to 10 per cent. The caloric value of a litre of mother's milk is 780, while a litre of cow's milk yields but 680 calories. An average four months old breast-fed baby takes about 800 grams of milk in twenty-four hours which is equal to about 625 calories. An artificially-fed baby on a percentage formula of fat 3, proteid 1.5, sugar 6.50 and taking 800 grams (27 ounces) in twenty-four hours receives only 470 calories.

The caloric value of the food is easily estimated from the total solids. Each grain of carbohydrate and of proteid has a caloric value of four and one-tenth, and a gram of fat gives a caloric value of nine and three-tenths.

The total solids can be obtained by evaporation and weighing or estimated from the specific gravity and percentage of fat. The specific gravity can best be ascertained by the lactometer. Some care must be exercised in the reading, and the temperature of the milk should be taken in order to calculate the result at a temperature of 60°F. In a paper read before the Medical Society of the State of New York I described the Richmond sliding rule, by means of which the amount of total solids can be obtained at a glance without indulging in mathematics. This rule has a movable slide in the centre of its upper surface. On this slide are two scales, one representing the specific gravity and the other the lactometer reading. On one side of the stationary portion a scale is placed indicating the total solids. One per cent.

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of total solids equals one inch and is divided into tenths. On the lower portion of the same side a temperature scale is marked from 32°F., to 80°F., with an arrow opposite the 60°F. mark. The rule is operated as follows: The lactometer reading and temperature of the milk have been obtained. Set the rule with the lactometer reading that has been observed, opposite the arrow at the 60°F. mark. For the true lactometer reading or specific gravity notice the point on the movable part of the scale opposite the temperature at which the milk stood. For example, if the lactometer reading was 30 and the temperature of the milk 75°F., you would set the lactometer reading, 30, on the 60°F. temperature line, and it would be seen that the 75 line corresponded with 32 on the lactometer scale, which is the correct lactometer reading. The percentage of fat in the milk will have already been determined. The movable scale is adjusted so that the arrow points at the percentage of fat on the scale. With the scale thus adjusted the line indicating the lactometer reading is placed against the required percentage of total solids in the milk. Suppose, for instance, the fat is 3½ per cent., the slide would be moved so that the arrow is opposite the 3½ mark. The specific gravity was found to be 32. Opposite 32 will be found 12.4 which would be the required total solids in the milk.

The Babcock formula has been greatly simplified and consists in dividing the specific gravity of the milk by four, and adding to this one-fifth of the per cent. of fat. For instance, the specific gravity of a specimen of milk is 1,030.8 and the fat is 4 per cent. One-fourth of the specific gravity is 7.7 and one-fifth of the percentage of fat is .8, so by the rule the total solids not fat would be 8.5. Adding the percentage of fat we get 12.5 as the total solids.

TABLE B.—TOTAL SOLIDS.

Number.	Specific Gravity at 60° F.	Total solids, Richmond scale.	Total solids, Babcock scale.	Total solids, actual weight.
1	1029.2	11.3	11.1	10.6
2	1023.8	9.7	9.5	9.8
3	1032.3	13.4	13.23	13.37
4	1030.5	12.2	11.94	12.4
5	1031.8	13.1	12.87	13.24
6	1028.5	10.8	10.6	10.33
7	1025.3	10.3	10.14	9.60

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Number.	Specific Gravity at 60° F.	Total solids, Richmond scale.	Total solids, Babcock scale.	Total solids, actual weight.
8	1025.	10.3	10.09	10.
9	1030.2	12.2	11.99	11.89
10	1029.	11.3	11.09	10.56
11	1027.	12.3	12.15	11.9
12	1028.	11.2	11.08	10.78
13	1032.	13.8	13.65	13.64
14	1032.2	13.	12.85	12.87
15	1033.2	13.	12.86	12.64
16	1031.4	13.1	12.89	12.96
17	1027.	11.	10.83	10.80
18	1029.8	10.7	10.5	10.15
19	1031.	12.7	12.5	11.69
20	1028.8	10.8	10.6	10.26
21	1029.8	11.7	11.5	10.83
22	1031.	11.5	11.35	11.36
23	1036.	11.3	11.1	10.96
24	1032.	11.8	11.6	11.75
25	1028.	11.7	11.56	11.43
26	1028.	11.7	11.56	11.44
27	1030.	12.	11.82	11.75
28	1031.	12.	11.84	11.75
29	1028.	10.1	9.8	9.46
30	1034.	13.1	12.9	12.55

The second table (see Table B) gives the corrected lactometer reading or specific gravity, and the amount of total solids computed by the Richmond and Babcock formulas. The amount of total solids obtained by slow evaporation and weighing is given for the purpose of comparison. It will be seen that the results obtained from the formulas are very similar to those obtained by weighing. The Babcock formula gives results slightly lower than the Richmond scale but the difference is very slight and the sliding-milk rule can be used for all practical purposes.

The amount of the proteids can be obtained approximately by subtracting the percentage of fat, sugar and salts from the total solids. The percentage of sugar in mother's milk averages 6.5 per cent., and rarely varies except in a small range. The average amount of the salts in mother's milk is two-tenths of 1 per cent.

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In cow's milk these figures are different. The sugar averages 4.5 per cent., and the salts, seven-tenths of 1 per cent.

For example, the total solids in a specimen of cow's milk amount to 12.4 per cent., and the percentage of fat is 3½, the solids not fat would be 8.9. Subtract 5.3 the sum of the sugar and salt percentage, and you would get 3.6, the amount of proteids.

While this is a rough method and throws all the error on the proteids, it gives us valuable information, which is reliable enough for ordinary modifications of milk.

DISCUSSION.

DR. ROTCH.—I think that this especial work of Dr. Edsall's is the most satisfactory that has ever been done in regard to the correctness of our results in modifying milk. It is the most judicial and the best exposition of the subject that I have ever heard; the ground which it takes is so absolutely correct. It is very gratifying to me that so much trouble should be taken to determine a question of this kind. We have arrived at the point now, I think, where the Society upholds the method of percentage feeding and, as Dr. Edsall has put it, if we can get it done at home as well and as accurately as at the milk laboratory, then have it done; and if not, have it done at the laboratory. I have never supposed I was getting absolutely accurate percentages when feeding by the percentage method but only closely approximate results. The system of home modification as evolved by Dr. Ladd has in my hands been very satisfactory.

DR. ADAMS.—Both of these papers contain, I think, some of the most valuable information that has been imparted to the Society in any discussion. The unreliability of milk mixtures is appalling and I have been repeatedly disappointed in percentage feedings. There are undoubtedly laboratory leaks, and very bad leaks but, as Dr. Edsall has explained, they are due either to inefficiencies or to carelessness. If we have a modifier who is honest then we can get nearer the accurate percentages from a reliable laboratory than we can possibly get from any domestic modification. I have had tests made of milk from the laboratory by various methods, and various tables have been compiled, and the unreliability of the abstraction of cream is, as I stated, most appalling. After estimating that an infant is getting a 3 per cent. fat, to be told that the child is getting between 6 and 7 per cent. is discouraging. At the hospital we have tried siphoning from

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above, siphoning from below, pouring the cream, and, the most reliable, the dipper method. The best dipper for use by the nurse or by the mother is the one with the conical bottom. In domestic modification I believe this will give the nearest to accurate percentages.

We have all been interested in the paper which was written and circulated very extensively by Dr. Wentworth, who has done good work in this line of investigation. I have had analyses made for me and I have gotten good results in twenty-three of the twenty-six analyses that were made. Lately I have studied Dr. Wentworth's paper carefully, and it seems to me that it is open to considerable criticism. In the first place, out of twenty-six of his analyses of laboratory milk, ten gave incomplete details in regard to control analyses. In twenty-three of the twenty-six analyses, in which control experiments are given in regard to the estimation of the fats and sugars, the percentage of sugar in the original specimen and in the control is said to have been estimated by polariscope, and yet, in each one of these 23 cases the percentage of sugar of the original specimen exactly corresponds to the percentage of sugar in the control, even to the second decimal place, a result which two leading milk analysts say they could not duplicate.

Furthermore, five specimens of milk were taken and the upper eight ounces of each were analyzed and compared with the lower twenty-four ounces. The analyses showed in every respect higher proteid percentages in the cream layer than in the lower portion, in 1 case being 1 per cent. higher in the condensed creamy layer than in the lower partially separated milk. The five specimens averaged 0.71 per cent. higher in the cream layer than in the milk. This is contrary to the theory and to the practical analyses of chemists. The fat in cream, by displacing the liquid plasm containing the proteids, necessarily lowers the proteid percentage of the milk. Dr. Wentworth quotes Hammarsten in explanation of this peculiar result of his analyses, but if the reference is looked up, one will find no authority for the statement. The analyses quoted are averages of many thousand specimens of milk compared with the average of many analyses of creams. They do not represent the analyses of the cream of one specimen with the bottom milk of the same specimen.

As Dr. Edsall said, there are many leaks, and I was thoroughly impressed with the remark of his that if the man in the laboratory is looking for a certain thing, he is pretty sure to find it, unless the many channels of error are obliterated. And so it is in analytic work if we make up our minds to find the error of somebody else. With Walker-Gordon Laboratories—if you want to use the term—we all know that they make errors, and some of them are bad ones; but there is undoubtedly more accuracy obtained by their skilled assistants than can be possibly gotten by any method of domestic modification.

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DR. COTTON.—I agree with Dr. Adams in the statement that these papers are of great importance, not so much in regard to the technique, but in regard to the findings. It has been a matter of a great deal of curiosity to us as to how accurate these mixtures from the laboratory might be, and as to how accurate our home modifications might be. The same work has been done along this line by others, but not to my knowledge has there been anything to compare with the work presented here by Dr. Edsall. As valuable as it may be, it is limited. We are not all chemists, and we cannot all afford to employ chemists to make these exhaustive analyses of milk; and yet, it is of extreme importance that we make effort, constantly, to determine our milk mixtures.

The manager of the Walker-Gordon Laboratory, in Chicago, gave me a hint and I found a little test tube, such as Dr. Shaw presented, supposed to be for work along the line of the Leffmann-Beam method. I was delighted because, as you know, in order to use the Babcock test at least 18 cc. of milk must be secured and many a breast will not yield that when you need it. This little Leffmann-Beam tube is very inviting, its capacity, the small amount required. There is difficulty in cleaning the little capillary tube, but by having a delicate, long pipette made to insert into the tube, the difficulty is obviated.

Professor Haines, of Chicago, at one time asked me if I could depend upon the method I was using. It does not make any difference whether your race track is $^1/_{16}$ of a furlong short or long, if you always use that track to speed your horses on; it does not make any difference whether your yard stick is a little longer or shorter, if you buy and sell by that nobody is wrong. And so it does not make any difference whether your method is accurate so long as you always use the same method. Of course, when an observer uses different percentages, then there is a difference.

I have been asked, "What do you learn from an analysis of breast milk?" Very little to be sure, but if the attendants upon disorders of the nursling will persist, we will finally collect data enough to make deductions.

I think a great deal of valuable information may be obtained from repeated routine analyses of breast milk. The value of analyses of milk mixtures and of cow's milk does not need any emphasis but analyses of breast milk I believe we ought to take up and encourage.

DR. MORSE.—It is rather difficult to reconcile the discrepancy in the results obtained by Dr. Edsall and Dr. Wentworth. It seems to me that there are two possible explanations: either that one of them was wrong, or that they were using different materials. My own feeling is that they were probably both right, and that the modifier in Philadelphia was doing better work than was the modifier in Boston the year before.

DR. HAMILL.—One thing in Dr. Edsall's results, which has

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interested me very much, is the difference in the fat percentage of the top milk from two of the dairies. Both of these milks are under the certification of the Philadelphia Pediatric Society Milk Commission. The results of the chemical analyses of the two milks have been fairly uniform; those of the one in which the cream rose poorly being a little evener than those of the other. When the top milk is removed from the former after it has stood for twelve hours, its fat content is found to vary in different samples from 7 to 12 per cent., while in the latter it is uniform. This result is suggestive. It is a well-known fact that the fat of milk is easily melted and that even mild degrees of heat will produce sufficient coalescence of the fat globules to interfere with the proper rising of the cream. I know of no other condition that would produce this result. Therefore, it is desirable that our Commission investigate the condition of this milk.

The three samples of modified milk which I gave Dr. Edsall for his analyses were prepared under entirely different conditions. The first one, which was the most accurate, was mixed by the mother of the child. She is the sister of a physician—not only a physician, but a Boston physician—and she had been instructed by her brother in the art of modifying milk. I observed her in the preparation of the mixture which Dr. Edsall examined, and I am satisfied that her methods were as accurate as we can hope to have them in home modifications. This mixture was made from whole milk and centrifugal cream of definite percentages from one of the certified milks. The second mixture was prepared from milk obtained from one of the best uncertified milks in Philadelphia. The results in this instance were not as good as in the former, but they were fairly accurate. The third mixture was obtained from a dispensary patient; she was a woman of fair intelligence, but she used for her modification a market milk of very unsavory reputation. The first bottle which she submitted soured before it reached the laboratory. A bottle of the whole milk used in the preparation of this mixture contained but 2.1 per cent. of fat. In the light of this result, I suggested a change of milk man. The mother told the new milk man her reasons for changing. He provided her with "a rich milk, especially used for babies." The mixture prepared from this milk, instead of containing 3.5 per cent of fat, as was desired, contained 5.1 per cent. The milk man had been true to his promise to provide a rich milk.

The work which Dr. Edsall has done is most valuable, and I agree entirely with his deductions, but I am not in accord with Dr. Morse in thinking that the difference in the results of the examinations of laboratory milks from those obtained by Dr. Wentworth, are due solely to the fact that the milks were prepared in different cities by different modifiers. This may have had some influence upon the result, but I do not believe that Dr. Wentworth's results have any claim to be compared with those which

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Dr. Edsall has just given us. Dr. Edsall and his assistant, Dr. Fife, are trained chemists. Dr. Wentworth does not lay claim to this distinction. The methods which he used are recognized methods of inaccuracy. The methods used by Dr. Edsall and Dr. Fife are those used today by all chemists of standing and the only ones by which accurate results can be obtained.

DR. CHAPIN.—The Babcock test is a thing I have worked with a great deal. Its chief advantages are that it can be quickly and easily handled by the average physician, and it is accurate.

I think that the future study of this milk question must be largely with the proteids. We have devoted a great deal of attention to the fats and now I think it is time to devote more attention to the proteids. Different observers do not agree as yet on the various methods of separating the proteids of milk. I have looked up the subject very carefully and I find among the best authors a great divergence of opinion as to their work and a brief paragraph from a paper I read last summer will explain what I mean.

"It will be evident that until the *composition* of the different proteid bodies found in milks is discovered, and some uniform methods of separating them are agreed upon, comparing milks by analyses made by different methods may lead to erroneous conclusions. Whey and cream mixtures may not be such close imitations of mother's milk as have been supposed. At present the comparison of milk analyses for proteid is of little use except by showing total proteid and thus the potential food value."

I think that this is a subject for further study by experts who are capable of doing it.

DR. EDSALL.—I used the ether extraction and the various forms of which I spoke in connection with the centrifugal method, because I wanted accuracy, so far as possible, in trying to settle the question. I do believe that the centrifugal methods that can be used clinically, are fairly accurate and are very useful for clinical purposes. One of the things I left for Dr. Shaw to say, and which he did say, is that it is extremely important that these estimations be made. As Dr. Cotton said we are all of us willing to make an attempt to make an accurate analysis in cases in which we are in trouble, and I think if pediatricians are interested in the accurate feeding of infants, they should be willing to learn something about the simple methods of determining whether it is right or not. Another thing that adds to accuracy: if fats are estimated by a centrifugal method, one must have a care in doing one thing, that is to make a correction of the specific gravity of the mixtures. It can be easily corrected by a table and takes only a moment.

I did not present my work in any special relation to Dr. Wentworth's work. I do feel, however, that Dr. Morse's suggestion may have something to do with the matter. It is possible that

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one modifier in one city is doing better work than a modifier in another city. But that does not explain all of it; there is a difference in methods.

We may not, of course, attempt in this series of estimations to make any separate determinations for casein and other proteids present in milk. Those things are entirely out of the line of clinicians, I think. It is very difficult to make accurate determinations, and if anything approaching accuracy is started for, it will take a great deal of time.

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DISCUSSION ON THE RELATION OF THE BACILLUS OF SHIGA TO THE SUMMER DIARRHEAS OF CHILDREN.

Dr. Simon Flexner, of Philadelphia, opened the discussion thus:—

"Mr. President and members of the Pediatric Society, I wish to express my great pleasure at the invitation extended by your officers to present to you my experience, gathered chiefly during the past summer, in respect to the bacteriology of the dejecta of the intestines in cases of socalled summer diarrheas, which may be of some use in clearing up at least the gravest of cases of this very important and hitherto very obscure disease. You all know, of course, that there has been no want of effort to discover the cause of these diarrheal diseases, but very little, if any, definite information has been gathered or had been gathered, with reference to the cause of diarrheal diseases, until the observations of which I shall speak. You are all familiar with the work of Escherich and his pupils, of Booker and other investigators in this country, and familiar with the fact that their results have left the etiology of the diarrheal diseases of infants without a solution. We have according to their results a number of possible etiological factors, chiefly bacteriological, present especially in cities, which may or may not be the causes of such disease. During the last few years the introduction of a slightly new method of approaching the subject of the bacteriology of certain forms of dysentery has met with such success in unraveling this complicated and very difficult disease in adults that it seemed that perhaps the application of the same method to the study of the bacteriology of the diarrheal diseases of infants might yield similar results, or results at least of value.

I became interested in the study of dysentery some three or four years ago, while on an expedition to the Philippine Islands. While there we had the opportunity of studying a considerable number of cases of acute dysentery in adults, and had no difficulty in establishing the existence in these cases, both in the dejecta and in the pathologically altered intestine, of a microorganism, perfectly well defined, differing from the ordinary intestinal bacilli which we are familiar with, an organism which had

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only a short time before been described as the cause of an outbreak of dysentery in Japan by the man whose name it bears, Shiga. Now, Shiga introduced a modification of the method that led to his success and to the success of others in the same field, to which, I think, all our progress is due. He conceived the idea that if, among the host of organisms that can at all times be cultivated from the diseased intestine, the organism causing the pathological lesion were there, the chances were that it would produce such changes in the blood as to cause agglutination and thus show its relation to this pathological process. He observed that by means of the socalled Widal test it was possible to pick out of all organisms which might be isolated in cultures from the stools in typhoid fever that particular one which probably is the cause of the disease. He worked, as I stated, with an epidemic of dysentery—these epidemics of dysentery in Japan were common occurrences—and as a result he discovered that in all cases a single organism was picked out by means of this agglutination reaction. When he came to study the properties of this organism he found that it differed from all the ordinary bacilli. Then he found that it agreed perhaps best with typhoid bacilli. It differed somewhat from the typhoid bacillus in certain points; these permitted its detection and separation from that group of organisms. I think now that this bacillus of Shiga has come to be recognized as the cause of a definite group of cases of dysentery in adults. Since that time his observations have been confirmed by the Johns Hopkins Commission which was sent to the Philippine Islands, by Kruse, of Germany, and indeed in the last few years by bacteriologists and pathologists wherever these cases have occurred and wherever bacteriological science is pursued. There is, therefore, an unanimity of opinion among them that there is a definite bacterial form of dysentery which differs from amebic dysentery and which is marked out as a definite disease. It was with these results in mind that I thought it worth while to turn to the study of diarrheal diseases in children, and I very early secured the cooperation of Dr. Knox, who was in charge of the Wilson Sanitarium near Baltimore, and of Mr. Bassett, and of Mr. Duvall, a bacteriologist who had had previous experience in the study of dysenteric diseases in this country, in which he had demonstrated the occurrence of the Shiga bacillus. It was possible to study at the Wilson Sanitarium something like 50 cases of the diarrheal diseases of infants. I cannot tell you

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anything, I regret to say, about the clinical data with reference to these cases; I am not a clinician and things which would appear to me probably would be very gross and of little use to you in the types of the disease in which this organism was found. Of the cases which came to the sanitarium, about 52 cases were taken just as they presented themselves, without any selection whatever, and studied with reference to the occurrence of this organism, and it was found that in, I think, 45 of the 52 cases, perhaps a few more, the bacillus of Shiga was present. It was also found that not only was this bacillus of Shiga present, but that the blood serum of children who had this bacillus in the intestinal mucosa reacted with the organism in question, and often with organisms obtained from the dejecta of cases of dysentery in the adult, with organisms which I had obtained in the Philippines, and with those obtained by Kruse and others. It seemed, therefore, that this much could be stated: In a large proportion of the cases of summer diarrhea which came to the Wilson Sanitarium during the past summer, or at least during some weeks in the summer, which were examined bacteriologically, there could be obtained this microorganism, which is, I think, pretty well established as the cause of a definite type of adult dysentery. The cases in which the organism was obtained were cases in which blood and mucus appeared in the stools. That much I can tell you; what the other clinical facts are I really do not know.

We found that the organism is not easy to obtain on account of its similarity to other intestinal organisms from formed stools or from feces. Our success depended largely upon the condition of the stools. If blood also occurred along with the mucus in the stools, the organisms were obtained with even greater ease than when mucus occurred with an absence of blood. So an appearance of mucus and blood in the stools led us to feel that the organism would always be obtained. That is about the status of the studies so far as they were carried out last summer, and that is the basis for the most part for any notion which may exist that the bacillus of Shiga is concerned with the causation of a group of cases of the summer diarrheas in infants. It was not possible to carry out these studies during the summer in other places. In Philadelphia we did not have good opportunities to study children in this way, but we did have a few cases. This is, however, no indication whatever of the number of cases which occurred there, but they represent all of the cases which we had access to.

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There are one or two points of great practical interest which I should like to bring to your attention. I feel that those interested in the bacteriology of the diarrheal diseases of children should undertake to discover whether the disease as it will appear this summer is the same; I should like to know what your experiences will be. I should like to speak to you as one who has had his difficulties with the isolation of the bacillus of Shiga in order that you may not draw too hasty conclusions as to the presence or absence of this organism. In the first place, I think it is desirable that those who are intending to study the bacteriology of the intestinal flora of infants suffering with diarrheal diseases should take advantage of the opportunity to familiarize themselves with the bacillus of Shiga before the studies are begun. It is, in fact, a very easy organism to cultivate, and it does not offer any very great difficulties in learning its properties; but the real difficulty comes from the fact that the organism is not so very different from the colon bacillus and from the typhoid bacillus, as observed under ordinary conditions, so that one who is not familiar with it might easily be led astray in his work and in his conclusions. So it seems to me that any work that is contemplated should be preceded by a study of the organism itself. I shall be only too glad to send cultures of the organism to any one who has a desire to study it.

Now there is another point. The mere knowledge of the organism is by no means an assurance that you will be able to pick it out of the mixture of organisms which always occur in the intestines. There are tricks in the trade of bacteriology perhaps, as in all other trades, and the knowledge of the tricks will facilitate very greatly the accomplishment of the purpose that you have in view. The trick which helps one very much in picking out this organism is the following one. If you make cultures of the dysentery bacillus and close up the plates, at the end of twenty-four hours you will have abundant growths of the dysentery bacillus, but if you mix colon bacillus and dysentery bacillus and, having sowed this mixture in plates, put these to devolop in the thermostat, you will find that the dysentery bacilli, when mixed with the colon bacilli, are considerably restricted in their development. If, therefore, you transplant at the end of twenty-four hours, you will get almost no dysentery bacilli and almost all colon bacilli. The thing to do is not to transplant any colonies after twenty-four hours, because the colon bacillus is so far ahead in

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its growth of the dysentery bacillus; and if you wait for an additional twenty-four hours you will not be much helped, because you will only have more colonies to confuse you. The trick is to take a blue wax pencil and mark out on each plate in a blue streak the colonies present at the end of twenty-four hours, and make up your mind that you will have nothing to do with them. Put the plates in the thermostat and then regard every colony that has come out after the first twenty-four hours as suspicious. You can tell at once by the blue streaks which of the colonies came out in the first twenty-four hours.

Now it is in the second group that you will most likely find the dysentery bacilli. You will find in some cases that the number of dysentery bacilli which are obtainable far exceeds the number of colon bacilli which have come out in the twenty-four hours. We had one such case in Baltimore last summer. There was practically a pure growth at the end of forty-eight hours and the colon bacillus was practically absent during that period. Often under favorable conditions in which you have blood and mucus in the stools, you transplant and get many suspicious colonies, perhaps eighteen or twenty; but in some cases you may get only one, two or three colonies of the bacillus of Shiga and many other colonies, colon bacillus, etc. I think I can predict that many of you will fail to find the organism even when it is present.

Then there are one or two other points which I might mention to you. As a result of the studies of Shiga and of the commission which went to the Philippines, and of Kruse in Germany, it was concluded that there were certain variations in agglutination. The studies of Dr. Hiss in this country, independent of those which have been carried on since by Dr. Park in New York and Dr. Gay in Philadelphia, and others, have shown us that the dysentery bacillus does not represent a single type but that it has group characteristics. There are two well defined groups of organisms. The one when grown upon mannite will not make any change upon the litmus. But the second type of the organism, which has all of the other cultural properties of the first, does produce a change in the litmus. They have certain relations, yet certain differences, so that each one retains in a way its certain individualities. We cannot say, if we admit that this organism is the cause of dysentery in infants, that one or the other type is especially concerned with the infection. We have found that the types occur somewhat irregularly, in an epidemic; we may find one type in one epidemic,

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another in another epidemic, that is in adult dysentery, and in some epidemics we may find both types of the bacilli occurring. All this indicates that the dysentery bacilli represent a group rather than a special or specific type of organism, just as we now recognize the colon group, etc., of organisms. It is of very great importance, both scientifically and otherwise, to have discovered these two types of organism, and it is furthermore important to know that these types vary. As we have stated, the particular type which has been obtained from cases of diarrheal diseases of children does not ferment with acid production on sugar, but both of them do on glucose and some on mannite. This year we have found one that agrees with the original Shiga type, but I do not think we have pursued this subject very carefully. These facts have come out since our study last summer, and it is very important that the investigations shall be taken up and carried out during the present summer. And I think it is safe to say that before the summer has gone we shall know, not only more about the diarrheal diseases, but also very much more about the types of the organism which are concerned in its production, whether it is one type or both types combined. Everything is left to the future, we have simply made a start. We have established the presence in these diarrheal diseases of an organism which previously had not been discovered in them. We have evidence of its pathogenic action. It is the future which must determine what position this organism must occupy with reference to the etiology of the summer diarrheas."

Dr. W. H. Park, of New York, spoke as follows:—

"My personal interest in the subject under discussion started with an experience last summer at Seal Harbor, Me., where Dr. Flexner and I happened to be visiting at a time when there was a slight epidemic of dysentery. At Dr. Flexner's suggestion, Dr. Dunham and I looked for and found that the mannite fermenting type of the dysentery bacillus was present. When I returned to New York, I found to my surprise that in towns along the Bronx River, just north of New York City, there had been about 500 cases of dysentery among a population of 30,000. The sick were mostly children, though some were adults. The patients had characteristic dysentery; pain, tenesmus, blood and mucus, some vomiting and other symptoms. The mortality was about 4 per cent. Both varieties of dysentery bacilli were present. Shortly after

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that a preliminary report of Dr. Flexner's assistant, Duvall and Bassett, on the bacteriology of summer diarrheas came out. The conclusions arrived at by them clashed with my ideas and stimulated me to look further into the subject. There developed a few points which may be of interest. First, the agglutination test in dysentery is not going to be as easy to interpret as in typhoid. When an attack of summer diarrhea occurs in a previously healthy child, we can depend upon the serum reaction, but I found that a 1-10 reaction is very common in children having chronic intestinal diseases when no dysentery bacilli have been present. In adults where there is a chronic or subacute disturbance of the intestinal tract and no dysentery bacilli, we frequently find reactions between the serum and the mannite fermenting type of bacilli, in dilutions of 1-50 and even much higher. In the Bronx epidemic I found the children there giving strong reactions in convalescence. After three months the strength of the reactions diminished greatly, proving the agglutinism to have developed in the blood because of the dysentery. It averaged 1-30 at first and fell to below 1-10. There are other bacilli besides those belonging to the dysentery group whose injection into animals is followed by the development of agglutinating substances which clump the dysentery bacilli. I found in a number of cases of chronic dysentery in adults and in several children such bacilli, which in cultural characteristics were almost identical with the colon bacillus. These bacilli occurred more often in subacute and chronic cases than in acute cases. In New York City, where we had very little dysentery, children having diarrhea not accompanied by mucus and blood did not as a rule give a serum reaction with the dysentery bacilli, and I do not believe the dysentery bacilli were active in these cases. The results might be quite different in a town, or even in a city, where there happened to be a great deal of infection. It seems to me this must have been the case in Baltimore. From my own slight experience, therefore, and from the reports of others, I feel that the dysentery bacilli do not occur in the large proportion of cases of summer diarrhea, and I should expect mucus and blood in most cases where the dysentery bacillus is an important factor."

Dr. Henry Koplik, of New York, continued the discussion:—

"I think that the subject of summer diarrhea is a very complex problem. Ever since the advent of modern bacteriology it has received extensive study. One among us, Dr. Booker, has made an

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international reputation in this field. In fact his studies on summer diarrhea will ever remain classical. He has met with microorganisms which, if they do not correspond exactly with what Dr. Flexner has described, certainly belong to the same group.

In considering the subject of summer diarrhea, we must first take into account that all practitioners who see any amount of summer diarrhea in children recognize certain broad definite classes of cases. In the first place, the great mass of illness in the summer time is found among artificially fed infants, and in that class we find a number of diarrheas which are the result primarily of the anatomical and physiological insufficiencies of the gut. We introduce into the baby's stomach a foreign element, that is cow's milk, and the gut is subjected to constant traumatism, as it were, from an anatomical and physiological standpoint. The first group of disturbances, which every one has met, is the dyspeptic group. This gradually then merges into other groups. If the milk is correctly modified or its administration suspended and something more digestible substituted, the digestive disturbances cease and the baby is practically convalescent. We must recognize this group because it is a very important one, and one that generally appears before other sets of symptoms belonging to the other groups of diarrhea make their advent. In other words, the dyspeptic attacks are due to an anatomical and physiological inability of the gut to cope with a foreign element, cow's milk. A great many of the severe summer diarrheas of infants are started in just this way.

The second group, I think, belongs to that class of diarrheas which are caused by the microorganisms in the food itself; that is, in the cow's milk, which, no matter in how cleanly a manner it is collected, carries with it a certain number of organisms which, if allowed to proliferate in this medium, will cause digestive or even inflammatory disturbances. This is a distinct class of cases, so distinct that its history reaches far back before the days of bacteriology. To this group would belong the cases described by Biedert, Escherich, and Booker due to the bacillus of butyric acid, in which there are a very considerable number of very foul smelling, acid stools, also those due to the bacillus lactis aerogenes. These form a very large group of cases in our dispensaries and clinics and by far the largest group of cases of summer diarrhea. They are the cases due to the bacteria in the food itself, indigenous in the milk.

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The third class of cases of summer diarrhea are the infectious cases. They are a distinct and independent class and have very little to do perhaps with the bacteria of the food itself. Some microorganisms, as Dr. Snow, has pointed out, have been introduced into the body from without. In this group belong the staphylococcus infections. Examining the dejecta of such babies, we have been struck by the peculiarities of the cases described as streptococcus infections by Booker and Escherich. The stools are peculiar; they have certain characteristics with certain clinical features.

Another set of cases belonging to this infectious group is the one which has been mentioned by Dr. Flexner, and this class has been the subject of investigation for the last four or five years. Ever since the discovery of Shiga, the pupils of Escherich and Escherich himself have been studying this set of diarrheas; the socalled bacilliary diarrheas. The bacilliary diarrheas occur for the most part in older children. They may occur in very young infants and they have been found to occur in institutions, or in certain localities. They are a distinctly limited class of diarrheas. How large this class is, future studies will show. Escherich and his pupils found in these diarrheas a bacillus which in its reactions exactly corresponds to the Shiga bacillus and which, according to Escherich, gave the agglutination reaction, as in Dr. Flexner's cases, but Escherich hesitated to say that the Shiga bacillus and this bacillus were exactly identical, or that his cases were true dysentery. Dr. Flexner in his remarks indicated why he himself had hesitated, because this is a class in which there are a number of bacilli which resemble each other closely. Dr. Park has shown you also that the agglutination reaction obtained in this class of cases is more or less uncertain. In my own service I had a number of cases last summer, and we have been trying to obtain some definite results in the etiology and bacteriology. In none of our cases could we be absolutely sure that we obtained the bacillus described by Escherich and his pupils and Flexner and his pupils. I do not say that Flexner's bacillus and Escherich's bacillus are identical, but they do certainly belong to the Shiga group. We are not ready to assume that this set of summer diarrheas belong to the dysenteries.

I have tried to mark out how complex this whole subject is. There is another point which I wish to dwell upon. I do not know that I stand apart from the rest of the pathologists and

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bacteriologists when I say that in some cases of diarrhea these bacteria combine to form a complex etiology; that is, diarrhea in a baby may be due to several causes. It is not always that you find the streptococcus or the staphylococcus the actual cause, pure and simple. As to this particular Shiga group I would hesitate to say at present how great the class is. I am inclined to think with Dr. Park and with Dr. Escherich that the group is a limited one. That it does not include all the forms that we see in large cities, that it does not take in the forms which I think are gradually being controlled by our modern methods of infant feeding among the poor from distributing stations, which give these little patients clean milk. I have received children under my care who I am sure would have died in the days preceding the agitation on sterilized milk. We have saved them, and they belong to the second class of cases in which the bacteria, indigenous in the milk itself, had been allowed to grow and get the upper hand. These bacteria were introduced into the intestinal canal of the infant with the decomposed milk and caused trouble.

The clinical and bacteriological study of summer diarrhea should go hand in hand."

Dr. L. Emmett Holt, of New York, continued the discussion, saying:—

"During the past season, from October till May, considerable work has been done in the Babies' Hospital upon the Shiga bacillus and the diseases in which it has been found. In the laboratory of the hospital, Dr. Wollstein has studied bacteriologically the stools from 112 children, in most of the cases a number of examinations being made. These cases were taken at random and included all kinds of intestinal disturbance. The Shiga bacillus was found in 37 cases. These 37 all had symptoms of colitis and most of them were acute, usually beginning with fever which frequently was as high as 103° F. The stools showed much mucus and often they contained blood. In no case where both blood and mucus were present was the organism absent, although it was not found in all the cases where the stools contained mucus, even though the quantity of mucus was considerable. Ten of these cases occurred at the Babies' Hospital, three at the Nursery and Child's Hospital, twenty-four at the New York Foundling Hospital. Rarely were isolated cases seen, in nearly every instance several occurred together in a ward.

Characteristic blood reactions were obtained in all the cases.

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These were irregular in their appearance during the first week, although they were obtained in one case on the second and in another on the third day of the attack. During the second week most of the cases gave positive results. The reactions were present in one when it was discharged from the hospital, twenty-seven days after the attack. Ten newly-born and ten healthy children were examined, and in no one was the blood reaction obtained with the Shiga serum. The bacilli persisted in the stools for a considerable time after the attacks; in 1 case they were found six weeks and another four weeks after.

Fifteen autopsies upon cases of Shiga colitis have been studied by Dr. John Howland. There were no characteristic anatomical changes which accompanied this form of infection. Nearly all varieties of inflammation were met with except the croupous form, which was not seen in any of the cases. In the 6 acute cases there was an intense congestion of the mucous membrane, with areas of necrosis more or less extensive. In the 5 subacute cases there was follicular ulceration which was usually superficial. In 2 cases of terminal infection only a slight congestion of the mucous membrane was present.

Flexner's anti-dysenteric serum was used in 8 cases. In 3 of these very striking improvement appeared to follow its administration. Two children were practically moribund when it was given; in 3 others no effect was seen.

The number of cases of course is too small to admit of any deductions. The conditions in the intestines are so complicated, it is doubtful whether we shall see the striking benefit following the use of diphtheria antitoxin. The close relationship between a certain type of inflammation of the colon, viz., that with blood and mucus in the stools, seemed clearly established by these investigations."

Dr. J. H. M. Knox, of Baltimore, spoke as follows:—

"I am exceedingly interested, as we all are, in this discussion, and I have learned a great deal. I had charge last summer of the Thomas Wilson Sanitarium and a word or two about the clinical records of the cases referred to by Dr. Flexner may be of interest.

The Thomas Wilson Sanitarium is about ten miles from Baltimore, and on that account it was difficult to obtain the children very early in the disease. This is a difficulty which Dr. Booker and others have always had in that institution; the children are

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usually sent to us pretty sick after they have been treated, for some days at least, in the city. Of the cases in which the organism was isolated, at least ten presented the acute symptoms recognized as those of fermentative or dyspeptic diarrhea. They had a history of an acute onset with fever, vomiting and diarrhea. The stools were first semi-fluid, then fluid; and finally, having lost their fecal character, became mucoid, the mucus being infrequently tinged with blood. In these cases, however, the blood was not excessive, only an occasional fleck in with the mucus; and in several of these instances, two or three at least, we found no blood at all, and the organism was recovered from the portions of the stool which were either clear or greenish mucus. A large number of these 42 cases I think belong to either the subacute or chronic type, in which the child came under our observation late in the disease, and the stools contained a great many leukocytes and more blood. There were only a few cases, however, in which the blood was excessive.

I would not assert that all of our cases, more than three hundred in number, of the socalled summer diarrhea were caused by this organism, but so far as could be told there was nothing either in the clinical history, the character of the stools, or the pathological condition at autopsy, to enable one to differentiate our cases from those recognized as typical epidemic summer diarrhea. I should like to corroborate what Dr. Holt has said in reference to the pathology of this condition. In several instances there were no gross lesions at all; the intestine was covered with an excess of mucus. There was no ulceration whatever, but quite a marked enlargement of the mesenteric lymph nodes, and a paie and rather thinned out condition of the mucosa. In other instances we found definite ulceration beginning, as he has said, with small ulcers on the solitary follicles. In none of the cases last summer did we find extensive ulceration, but the cases were clinically like those of the preceding summer, in some of which we found very extensive ulceration associated with marked thickening of the whole wall of the large intestine. I think it is important to emphasize the fact that the cases which were sent to the sanitarium came from all sections of the city, that is, they formed a part of the usual epidemic of diarrheal disorders annually recurrent in large cities during the warm months. Furthermore, we had blood sent to us from other cities from babies similarly affected, and it agglutinated the organisms isolated from our

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cases. In several cases seen outside the sanitarium, either the organism was obtained from the stool, or the blood agglutinated the Shiga bacillus.

I feel, therefore, that while it is necessary to have this work confirmed, we can at least be hopeful that a fairly large group, both of the acute and chronic cases of summer diarrhea will be found to be due to this organism."

Dr. Booker, of Baltimore, then spoke, as follows:—

"It has been so long since I have worked in the laboratory that I have almost forgotten my own work, and feel somewhat embarrassed in returning to the subject. I cannot speak from personal experience in regard to the bacillus separated by Duvall and Bassett from the diarrheal feces of infants last summer. There are many points to be considered and problems worked out before the relation between this bacillus and the summer diarrheas of infants can be determined. Whatever the relation may prove to be, the separation of the bacillus from the colon group, and the careful study which has been made of it by these two young men, is of great scientific interest, and may be regarded as one of the most important and satisfactory works that has been done in bacteriology in this country.

When I left the laboratory study of the summer diarrheas of infants I had come to the conclusion that it was a very complex subject, comprising a number of affections, in which most of the organs of the body are often implicated, and in which many different varieties of bacteria are concerned, the most important of which belong to the streptococcus group.

I have isolated between thirty and forty varieties of bacteria from the intestinal contents of infants affected with different forms of summer diarrhea, and at the first meeting of this Society, in 1889, I described among other bacteria, the separation of seven varieties of the colon group. From the morphology, cultural resemblances, and especially the milk reaction, the variety which I described as bacillus M appears identical with the bacillus of Duvall and Bassett. At the following meeting of the Society, in 1890, I reported a case of ulcerative catarrhal dysentery in which special attention was called to a bacillus found in large quantity in the diseased colon, which closely resembled the colon bacillus, but differed from it in not coagulating milk or reducing litmus.

Owing to the large number of varieties of bacteria upon which I was at work, and the limited means at my disposal for animal

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experimentation, the pathogenic properties of only a few of them could be tested. Some of these which were found in large quantities in the intestinal contents proved strongly pathogenic to lower animals. Other workers in this field have similarly found a number of varieties of bacteria, possessing pathogenic properties, in large numbers in the diarrheal feces of children. These considerations together with the manifold variety in the clinical features and anatomical changes in summer diarrheas speak against a specific excitor for these affections.

The Shiga bacillus is said to have been found by Duvall and Bassett chiefly in cases having mucus and blood in the stools. This does not give us much information as to the condition of the intestine. It is important to distinguish between clear mucus and muco-pus, and pure blood and blood mixed with pus in the stools. Pus is the ingredient in the stools which indicates ulceration of the intestine. Mucus is secreted and poured into the intestinal canal in large quantity. Under normal conditions so much of this is reabsorbed that only an imperceptible quantity passes out with the feces. When mucus appears in the stools in clear lumps it may be the result of very slight abnormality in the intestine, without any pathological lesions whatever. It is when mucus is clouded by leukocytes that it represents a serious condition of the intestine. So with blood in the stools. When pure, even in considerable amount, it may not signify a serious condition of the intestine. It is when blood is persistent, and especially when mixed with pus that it should cause apprehension.

The only case that came under my observation last summer in which examination was made for the Shiga bacillus belonged more to the cholera infantum type than the dysenteric. The child had been sick only a few days, and had not lost much flesh. It was slightly toxic, and had frequent watery stools, with very offensive, putrid odor, and without mucus or blood. From the condition of the child and nature of the stools, I felt confident that cultures would show large numbers of proteus vulgaris. Contents from the rectum were taken with a glass tube and delivered to Mr. Duvall within a few hours. He reported the cultures as almost pure of the Shiga bacillus and containing no proteus bacilli.

Two injections of Flexner's serum were given to the child. A very slight improvement was noticed after the first injection, but a more decided change for the worse followed the second injection, and the child died a few days later.

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As to the serum treatment of the summer diarrheas, we should not be too sanguine in our hopes. Even if the Shiga bacillus should prove to be the important exciting factor of these affections, as has been so confidently claimed for it, we cannot exclude the participation of other bacteria in the disease. It is also well to remember that we often have serious pathological lesions, deep and extensive ulcerations of the intestine, and more or less important changes in other organs of the body to deal with. Care should be used in estimating the effect of serum upon the disease. In no other disease does recovery so often happen from apparently hopeless conditions, and under most diverse methods of treatment. Seven years ago I used Marmorek's streptococcus serum in a number of cases of streptococcus enteritis. Decided improvement followed the use of the serum in every case except one, but it was only temporary and all of the cases died. In 1 case in which streptococci were the predominating microorganism in the stools, the improvement after injection of the serum was startling. The child was twenty-one months old, and had been sick with enteritis four or five weeks. It was in a state of coma with small, feeble pulse, which could scarcely be felt, and had cyanosis over much of the body; 10 cc. of Marmorek's serum was injected under the skin about 10 A.M. In the afternoon the child had regained consciousness, had a fair pulse, the cyanosis had completely disappeared, and the child was able to take the bottle. The improvement was of short duration, and the child died in spite of repeated doses of serum.

In the use of Flexner's serum, I think, if we get results from it, it will be when used in the early stage of the disease, before serious anatomical changes have occurred, and other organisms are brought into participation. It has been suggested that the rôle of the Shiga bacillus is to render the conditions in the intestine more suitable for the activity of other bacteria. If this should prove true, it is still more important to use the serum in the early stage of the disease."

Dr. Flexner closed the discussion:—

"I wish to thank the members of the Society for the very full way in which they have discussed this condition. I have been myself very much helped by the facts and the views which they have stated. I quite agree with the gentlemen who dwelt upon the complexity of the problem, for it is by no means easy. I

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thought I had made myself clear that I am not committed to any view of the causes of summer diarrheas, excepting that this organism occurred in some of them. It remains for the future to determine just what the limits of this occurrence are, what the importance of this occurrence may be, and how the pathology and bacteriology may be brought into harmony. I do not think that one can speak positively upon that subject now. I think the autopsy records add very greatly to our knowledge of the character of the lesions associated with this disease. Our experience at the Wilson Sanitarium was so limited that it hardly seems worth speaking of; but with the reports now of fifteen autopsies we see something of its possibilities.

There are two points which I should like to touch upon briefly. The first is that I am convinced that whatever the Shiga bacillus does, it does early. We have to deal with mixed infections, and how much of this complex mixture is a condition of an early infection is a very important question. How much damage the streptococcus and staphylococcus, which are constantly present, can add to the effects of the other bacilli, I think, is a point that should be kept in mind. The serum is, of course, experimental. It must be remembered at the outset that this serum attempts to deal not with a disease, the effects of which are due to a poisoning, as is the case with diphtheria; hence it is not a serum which acts as a therapeutic agent alone. What we have to deal with in this disease is an organism, and our serum must be able to attack the bacteria themselves and bring about their destruction. We have a recent communication from Kruse, who has used the serum in adults in Germany, with excellent effects, he thinks. What this serum will do in the cases of children is a matter to be settled by experience. But its good effects are likely to be gotten if it is used early. Of course, in dealing with a large number of cases, many of them would have gotten well without it, but if you wait until the symptoms are more severe, the outcome is doubtful. As Dr. Booker said, the serum for hopeful therapeutic effect should be given early. The serum as tested on animals is very effective. It rescued guinea pigs from infection almost without exception. I think it is at least worth trial, but whether it is to be used or not is to be decided by bacteriologists and clinicians."

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CONGENITAL GASTRIC SPASM.

BY J. PARK WEST, M.D.,

Bellaire, O.

Doubt has been expressed by some as to the existence of a true congenital hypertrophic stenosis of the pylorus. An examination of the literature and of some stomachs shown as specimens of this condition shows that two classes of cases have been described under this title. A few, a very few, cases are reported where there has been found postmortem hyperplasia of the epithelium, an increase of connective tissue in the submucosa, and of fibrous tissue in the muscular coat, in addition to hypertrophy of both the longitudinal and circular muscular layers, the presence of this abnormal tissue causing, sooner or later, a true stenosis. More cases are to be found, similar to the one here reported, where there is only found an hypertrophy of the circular muscular layer, the stenosis which undoubtedly exists being due almost entirely to spasm of the hypertrophied muscle. To the first class the title should be restricted, and to the second is better given the name suggested by Dr. John Thomson, viz.: congenital gastric spasm.

The practical importance of this distinction is lessened somewhat by the difficulty in differentiating the two conditions during life, as the symptoms are similar, and both may have a palpable tumor, gastric peristalsis, and dilatation. A few cases of congenital gastric spasm have been relieved by careful diet and treatment and others have had periods of relaxation of the spasm, but it is not to be expected that cases of true hypertrophic stenosis will be permanently benefited except by surgical procedures. Since the prospect that the cases of gastric spasm will be permanently relieved by treatment or that the relaxation will remain is very small, it would seem best that all cases of both classes should be submitted to an early surgical operation, and especially so if a palpable tumor is present.

History of Case.—The stomach here presented was taken from the body of Infant Dodd, who died when thirty-two days old. With the exception of the maternal grandmother, who died with typhoid fever, the grandparents are living and well. The

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father, thirty-eight years old, the principal of a school, has had symptoms of pyloric spasm at times for the past four years. The mother, twenty-four years old, is not very strong and is rather nervous. During the last half of this, her second pregnancy, she suffered a great deal with burning in, and eructations of a sour fluid from, her stomach. The only other child is a son three and one-half years old. He had colic severely and was very constipated for his first three months. Ever since then his bowels and appetite have been very irregular.

The baby, a male, was born July 12th after a normal labor and his estimated weight was ten pounds. On the next day his bowels and kidneys acted well. During the third twenty-four hours he had from fifteen to twenty large thin stools. This diarrhea stopped suddenly without apparent cause.

(1) *Vomiting.*—The child nursed vigorously within a half hour after his birth. The flow of milk was well established on the third day. There was retching on the first day, more on the second, and on the third day vomiting began and continued more or less until the death of the baby. The milk was usually expelled within five to thirty minutes, but a few times was retained for two or three hours and three or four times for four or five hours. The vomiting was always projectile and usually came on without warning, seldom being preceded by retching. Occasionally there was severe retching without vomiting. During the first part of the second week, when he was allowed to nurse all he wanted, he would often vomit before he was through nursing and immediately nurse again. The length of time he nursed seemed to have no relation to the length of time the milk was retained. On the thirteenth day the attending physicians made a diagnosis of ileocecal intussusception and indistinctly felt a tumor about two inches long. For the next three days (July 14th, 15th, 16th) he was given nothing by the stomach but an occasional spoonful of water and while there was no vomiting there was a great deal of retching. For these three days rectal feeding and injections were resorted to but without improvement in the symptoms. For the thirty-six hours following the three days' fast he was allowed to nurse for two minutes every two to four hours; and, while he would vomit within two minutes to two hours, it was thought he retained more of the milk than formerly. The appearance of the vomitus varied with the length of time it was retained in the stomach, that retained the longest being the most

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digested. What I saw in a bottle brought to me was one-third soft milk clot and two-thirds light, straw-colored liquid. At no time was it stained with bile but contained some mucus toward the end and during the last week was a very dark brown color and of an offensive odor.

(2) *Stools.*—After the diarrhea on the third day there was obstinate constipation. A rectal injection was given at least once each day that usually caused a slight movement, and occasionally there was a very small passage without the injection. Four passages were shown to me; in the four there was about two teaspoonsful, nine-tenths of which was green mucus, the other one-tenth being small yellow flakes irregularly distributed through it. Thirty-six hours after his fast he had a small stool very much like a milk stool, only not so bright a yellow, and two similar ones the next day. Otherwise they were as first described until the last week when the few were the color and consistency of tar and very offensive. At no time was there a bloody or blood-stained mucous stool.

(3) *Pain.*—The suffering of the baby was intense much of the time. The pain would come on after nursing, with vomiting and attempts at vomiting, and even the eructation of gas was painful. A teaspoonful of hot or cold water would excite it. From the first there was pain following nursing, and in addition severe paroxysms, often lasting a half hour, that were more severe after midnight. After a few nursings on the seventeenth and eighteenth days there was little or no pain.

(4) *Physical Examination.*—I saw the child with Drs. Wood and Gregg, the attending physicians, on the twenty-second day. It was considerably, but not greatly, emaciated. For the previous twenty-four hours it had been having small doses of opium and belladonna and was quiet. There was but slight distention of the abdomen, limited to the upper part and most noticeable in the left half of the epigastrium. Very slight gastric peristalsis could be seen at times but this was never prominent. Pressure on the stomach would cause a light wave of contraction and at long intervals this was felt without pressure exciting it. Standing at the side of the mother while the child nursed one could hear loud gurgling that continued, gradually growing fainter, and ceased when nursing ended. This could also be heard when there was vomiting or retching and continued throughout the illness, being more marked the last week. The abdominal walls were very soft

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and a swelling about one by one-half inch was easily felt one inch to the right of the median line and three-fourths inch below the border of the ribs. Nothing else abnormal was found.

General Symptoms.—The urinary secretion was high-colored and somewhat diminished but not scanty, owing, no doubt, to rectal injections. There was no fever until the last week. The child emaciated rapidly the last few days and died on the thirty-second day after having convulsions for seventeen hours. Its estimated weight at death was five pounds.

Autopsy.—This was limited to the abdomen. The stomach was full and contained some fluid and mucus. At the pylorus was found a very hard swelling one inch long and three-fourths inches in circumference. The cardiac end of the stomach (about one-third) was thin, distended, and prominent. Toward the pylorus (two-thirds of the stomach) the walls were thick and contracted. The examination of a section of the pylorus by the microscope shows only an hypertrophy of the circular muscular layer.

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A CASE OF CHONDRODYSTROPHY FETALIS.

BY J. PARK WEST, M.D.,
and
W. O. S. PIPER, M.D.,
Bellaire, O.

The following history is that of a typical case of chondrodystrophy fetalis and corresponds to "Type D" of Ballantyne's fetal bone diseases in his "Antenatal Pathology." Since attention has been called to this condition again quite a number of cases have been published. In addition to this case there have been in our vicinity five of these dwarfs; two of them, now adults, were born in the county. Another, now in Bellaire, a woman born in Germany about sixty-five years ago, does a thriving business as a fortune teller, and has a family of normal children and grandchildren.

A CASE OF CHONDRODYSTROPHY FETALIS FOURTEEN MONTHS OLD.

Baby Wilhelm was born in Bellaire, August 16, 1901, and the examination of these data is based on those made when she was fourteen months old. She is the third of three children, all females. The oldest died when six months old, with pneumonia; so far as we were able to ascertain she was a healthy child. The second is three and one-half years old and healthy. The grandparents are all living and well. No child like this has been known in either family. The parents were both born in this country. The

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father is forty years old and is very tall and slender. The mother is twenty-five years old and is slightly below the medium height. She had no sickness, unusual worry, or privation during her pregnancy. The first stage of labor was seven hours in duration, the second but a few minutes, and in every way normal. There was no hydramnios.

The child was very fat and chubby at birth and two peculiarities were noticed and remarked at the time. The first was the large round head that showed no effects of its passage through the birth-canal. The second was the marked shortness and thickness of the hands and fingers.

She has always been nursed by the mother, has grown very slowly, and from her birth has been very cross. She cut her two lower central incisors at the eighth month and the four uppers at the twelfth month and none since. When five months old a slight angular curvature of the lumbar spine was noticed. Since she was eight months old she has occasionally sweat about the head. Her only other sickness has been three short attacks of summer bowel trouble, not severe. The heart and lungs are normal, and her mind differs in no way from that of an ordinary child.

From her twelfth to her fourteenth month she was under the care of Dr. Piper when she was seen in consultation by Dr. West. A look at her head suggested rickets; the rest of her body suggested cretinism; a careful examination and a comparison with a normal child of the same age shows it to be a case of chondrodystrophy fetalis.

Although the child is quite thin she has a general stumpy appearance and the extremities look stunted. Her head is very large (19⅝ inches) and covered with a full growth of soft hair. The anterior fontanel is open, slightly depressed and measures 2x2 inches. The frontal suture is widely open and the saggital also. but to a lesser extent. No craniotabes. The frontal and parietal eminences are prominent, and the forehead and sides show but little bulging. The face is likewise large and is proportionate with the head. The bridge of the nose is very much depressed and the point turned up. There is slight exophthalmos. The ears are very large and well formed. The mouth is large and closed and the lower jaw rounded. The tongue is of the usual size and not protruded. The neck is extremely short and the sternum set very high. The thyroid cannot be felt. On each side of

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the neck, in each axilla, and on the left arm, are pads of soft fat 1x½x½ inch in size. The chest has a rather square formation, the sides being almost straight and the sternum sloping but little. There is no beading of the ribs. The abdomen is not unduly full but looks very long. The liver extends 1 inch and the spleen ¼ inch below the border of the ribs. Except for a kyphosis of the five lumbar vertebra (noticed at the fifth month) the spine is straight. The body looks long for the size of the child and seems, on account of the high position of the sternum, to be continuous with the head. There is about two and one-half times as much skin as the baby has use for. This superabundance is especially shown on the extremities and lower half of the body where the skin is loose, thick, soft and of a dull muddy color, and not at all irritable. In other places it is not quite so plentiful, thinner and irritable, slight rubbing producing an erythematous blush. There is no hair on the skin. The muscles are thin and soft. At the middle of the arms, at the ankles, and an inch above the ankles are depressions encircling the limbs as if a string was tied about them. The joints are not enlarged nor so prominent as usual in a thin baby and the motions are not restricted. The bones of the extremities are short, as the measurements will show, and there is almost no curving. The bones are thick, feel unusually hard, and the lines and prominences sharply marked. For instance, the external ridge of the fibula is very distinct, running up to a styloid process that reaches higher than the head of the tibia. With the arms extended along the body, the distal end of the palms reach only to a point opposite the umbilicus. When she is lying quietly her legs and feet turn out, the feet resting on their outer border. She uses all her extremities well but has never tried to put any weight on her legs. The feet are "pudgy and mole-like." The hands are short and stubby; the short, thick, pointed fingers were so noticeable at birth as to have attracted the attention of the family; this is partly due to the lax redundant skin, and, to a larger extent, to the pronounced thickening of the bones, especially of the phalanges. With the hand closed there is no arch at the metacarpo-phalangeal joints, the metacarpal bones appearing of the same length. The hand of this child presents another peculiarity shown very distinctly in the photograph. Instead of the fingers lying together the index and middle fingers point to the radial side of the hand while the ring and little fingers point to the ulnar side. This was first pointed out by Dr. John Thomson and is considered by

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him and others who have studied the condition to be pathognomonic of chondrodystrophy fetalis. (See cut.)

This child was seen and examined six months later, when she was twenty months old. Her body had grown ½ inch; her extremities none at all. Her weight increased 4¼ pounds. She had cut seven more teeth, now having thirteen. The fontanel has not closed any more and the kyphosis has not changed. She makes no attempt to sit alone and rarely tries to change her position. She has ceased being so cross and lies contentedly most of the time. Otherwise her condition and appearance is as described above.

So far no treatment used has been of any special benefit. Dr. Piper had her on thyroids and cod-liver oil for two months. The addition of suprarenal gland, a regulated diet, bathing, and fresh air made no improvement.

The main points wherein this child differs from a rachitic and from a normal child are in the length and form of the bones. Compared with a normal child of the same age this one is 5½ inches shorter from vertex to sole, ½ inch shorter from top of sternum to pubes, ½ inch from vertex to anterior superior spinous process, and 5 inches from anterior superior spinous process to sole. From the acromion to wrist it is 2½ inches shorter, the hand and wrist 1 inch shorter, the thigh 2¾, and the leg 3 inches shorter. The clavicle differs from the other long bones in volume and length; it is long and thin and causes a marked projection of the upper bone of the sternum. The bones are thickened, are distinctly hard, and the anatomical landmarks as distinctly made out as in a thin adult, thus giving one the impression of a small matured bone. They do not have that uniform smoothness of the rachitic bone, nor have they lost the normal form but rather have gained the adult form thus early. The circumference of this child's head is 1 inch greater than the other's but the diameters of the two are about the same. The shape of the head (which is not typically rachitic) and the sweating of the head are the only rachitic features present. There is only that beading of the chostrochondral joints to be felt in every thin child and there is no enlargement of other joints. The size of the face is proportionate to the head, both being large, and not the large head and small face of rickets.

A number of papers on this subject state that two breeds of dogs, viz.: dachshunds and bassets, are examples of this disease.

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I have here the bones of the legs of a dachshund that show this statement, at least in this particular instance, to be a mistake. Dr. Carl O. Folkens, of Cleveland, O., who breeds dachshunds, kindly sent us a healthy, half-grown thoroughbred specimen that the bones might be shown with this paper. In most of the bones, during their preparation (boiling without chemicals), the epiphysis and diaphysis became separated, in others they were easily taken apart, while in these where they are still adherent they can be separated with ease. There is in none of the long bones a premature ossification of the epiphyseal cartilage nor an ingrowth of the periosteum. You will also notice that instead of being heavy and dense the bones are light and thin; this is especially noticeable in the long bones that have been sawed.

MEASUREMENTS OF CHILD WITH CHONDRODYSTROPHY.

	Chondrodystrophy.	Healthy.
Length	24½ inches.	30 inches.
Weight	13¾ lbs.	21½ lbs.
Neck (circumference)	8 inches.	8 inches.
Chest (at nipples)	14 "	17½ "
Abdomen (at umbilicus)	15 "	18 "
Pelvis (at crests)	12½ "	16 "
Head:—Circumference	19⅜ "	18½ "
Ear to ear	11¼ "	11 "
Glabella to occiput	14 "	13 "
Diameters:—Bitemporal	4½ "	4¾ "
Biparietal	5¼ "	5¼ "
Antero-posterior	6½ "	6½ "
Glabella to occiput	6 "	5½ "
Vertex to ant. sup. spinous process	15½ "	16 "
Ant. sup. spinous process to sole	9 "	14 "
Sternum to pubes	10½ "	11 "
Sternum to umbilicus	8 "	7½ "
Umbilicus to pubes	2½ "	3½ "
Trochanter to condyle of femur	3¾ "	6½ "
Fibula	3¼ "	6 "
Tibia	3 "	6½ "
Foot (instep 4½ and 4¾)	3½ "	4½ "
Acromion to wrist	6½ "	9 "
Humerus	3 "	4½ "
Ulna	3 "	4¾ "
Hand and wrist	1¼ "	2¼ "

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A CASE OF PULMONARY OSTEOARTHROPATHY.

BY THOMAS MORGAN ROTCH, M.D.,

AND CHARLES HUNTER DUNN, M.D.,

Boston.

E. T., a boy, three years and seven months old, entered the Children's Hospital, on March 19, 1903.

FAMILY HISTORY was negative.

PERSONAL HISTORY.—He was born at full term, was healthy at birth and was breast fed. He had had measles and pertussis, and, with the exception of these diseases, had never been noticed to have had an acute febrile attack. He never had any pains or convulsions, and never had any trouble with his joints or limbs. The child sat up at ten months, but did not walk until he was two years old, and had always been rather backward.

He was sent to the hospital for continuous cough. For a year he had coughed, on an average, two or three times an hour, and for a month previous to his entering the hospital the cough had grown worse. At times he had been raising a yellowish sputum. He had had no night sweats, and had not lost in weight. The appetite was good and he did not have any digestive disturbance. The bowels had always been regular. For nine months previous to entering the hospital the parents had noticed an enlargement of the finger tips.

The physical examination showed him to be well developed and nourished. Skin of a good color. Did not speak plainly. Head rather more square than normal, fontanel closed. Twenty teeth. Tongue normal. Examination of the lungs showed flatness on percussion over the entire left front of the chest. There was marked dullness over the back of the left chest, but no absolute flatness. Throughout the left side of the chest, front and back, there were loud bronchial breathing, bronchophony, and increased tactile fremitus, and there were numerous crackling râles. Examination of the heart showed the impulse in the fifth left interspace in the mammary line. The cardiac area was not enlarged. The cardiac sounds were regular and of good quality. A systolic

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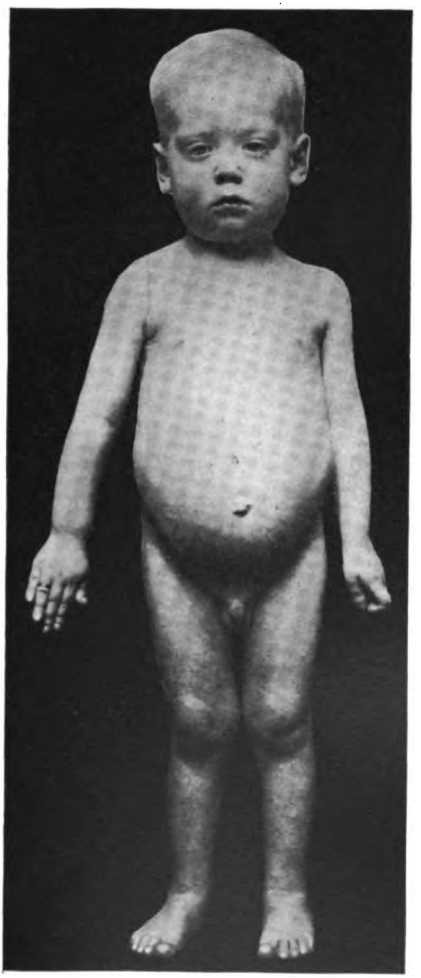
murmur was heard over the entire precordia, loudest at the second left interspace. The pulmonic second sound was not notably accentuated. The abdomen was very prominent and tympanitic, but there was no tenderness, and nothing abnormal was detected. The spleen was palpable just beneath the costal margin. The liver was not enlarged. There was no notable enlargement of the lymph nodes. The reflexes were normal. There was a marked enlargement of the tips of the fingers of both hands, giving a flat club-shaped appearance, and this enlargement was also noticeable in the distal phalanges of both great toes. A slight rosary was present, but no special enlargement of the epiphyses. Examination of the blood showed: Hemoglobin 70 per cent.; leukocytes 9,600; erythrocytes 4,320,000. A differential count of 500 leukocytes showed: Polymorphonuclear cells, 68 per cent.; large basophiles, 4.8 per cent.; small basophiles, 23 per cent.; eosinophiles, 2.6 per cent.; mast cells, 1 per cent.; myelocytes, .4 per cent. During the count there were seen no erythroblasts. The erythrocytes were normal except for a slight achromia.



PHOTOGRAPH OF THE PATIENT: A CASE OF PULMONARY OSTEOARTHROPATHY.

Examination of the urine showed: Color normal; reaction

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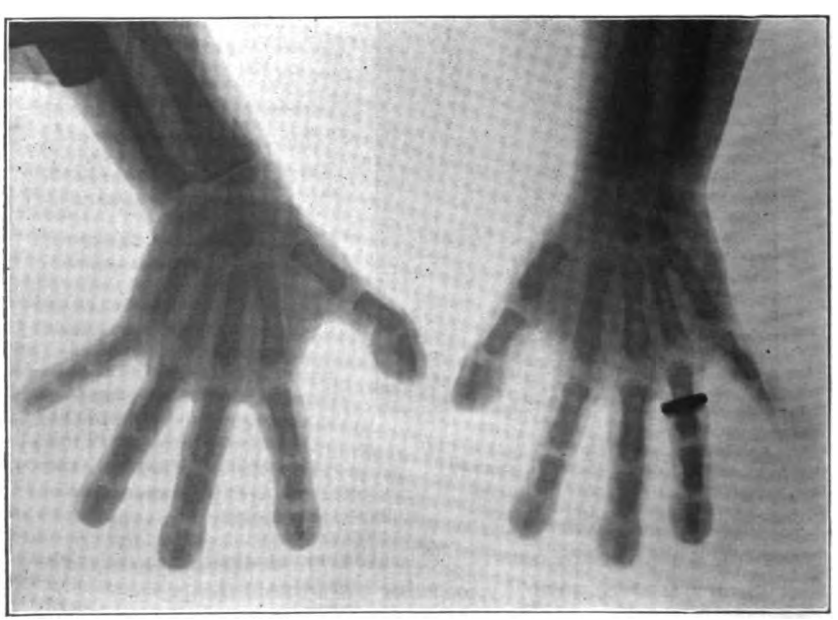
PHOTOGRAPH OF THE HANDS IN A CASE OF PULMONARY OSTEOARTHROPATHY.

acid; sp. gr. 1,023; urates increased; albumin slight trace; no bile and no sugar. Sediment small in amount and showing an occasional small round cell and a rare hyalin cast.

Examination of the sputum on March 19th, showed it to be



RADIOGRAPH OF THE HANDS IN A CASE OF PULMONARY OSTEOARTHROPATHY.

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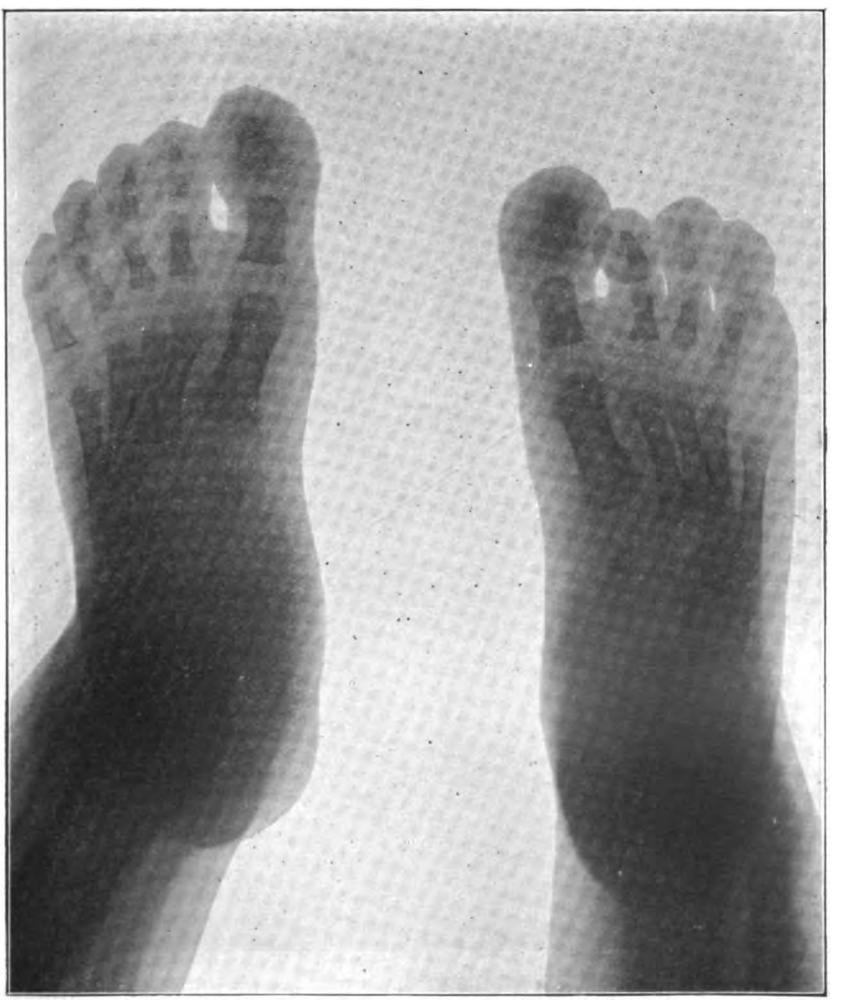
purulent and to be loaded with small bipolar gram-decolorizing bacilli, both intra- and extra-cellular. Culture made on blood serum, and on plain agar gave a few colonies of gram-staining



RADIOGRAPH OF THE FEET IN A CASE OF PULMONARY OSTEOARTHROPATHY.

cocci, but no bacilli. A culture on blood agar gave numerous, very small watery colonies. Cover glass preparations from these colonies showed small gram-decolorizing bipolar bacilli. (Pfeiffer's bacillus of influenza.)

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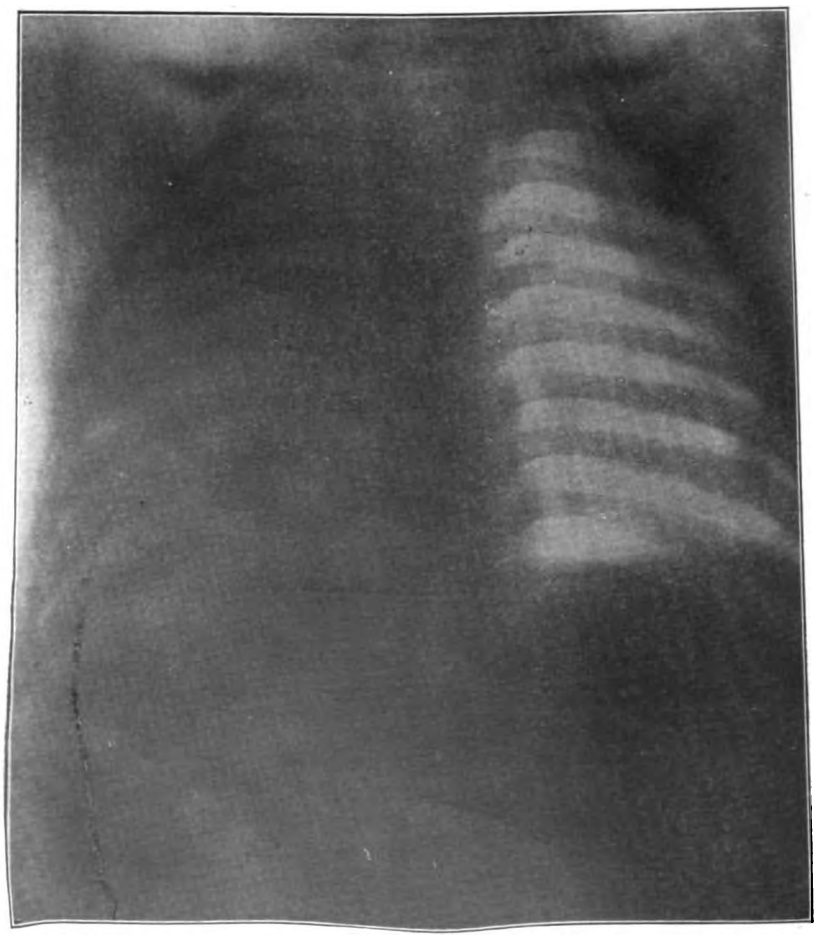
Thoracentesis on the left side showed no fluid, but a solidified lung. A tuberculin test was made on March 25th, and again on March 28th, with negative results.



RADIOGRAPH OF THE CHEST IN A CASE OF PULMONARY OSTEOARTHROPATHY SHOWING CONSOLIDATION OF THE LEFT LUNG.

During the following two months the child had coughed less and there was no expectoration, the physical signs remained unchanged. (See chart.)

On April 11th, sputum obtained from vomitus was examined

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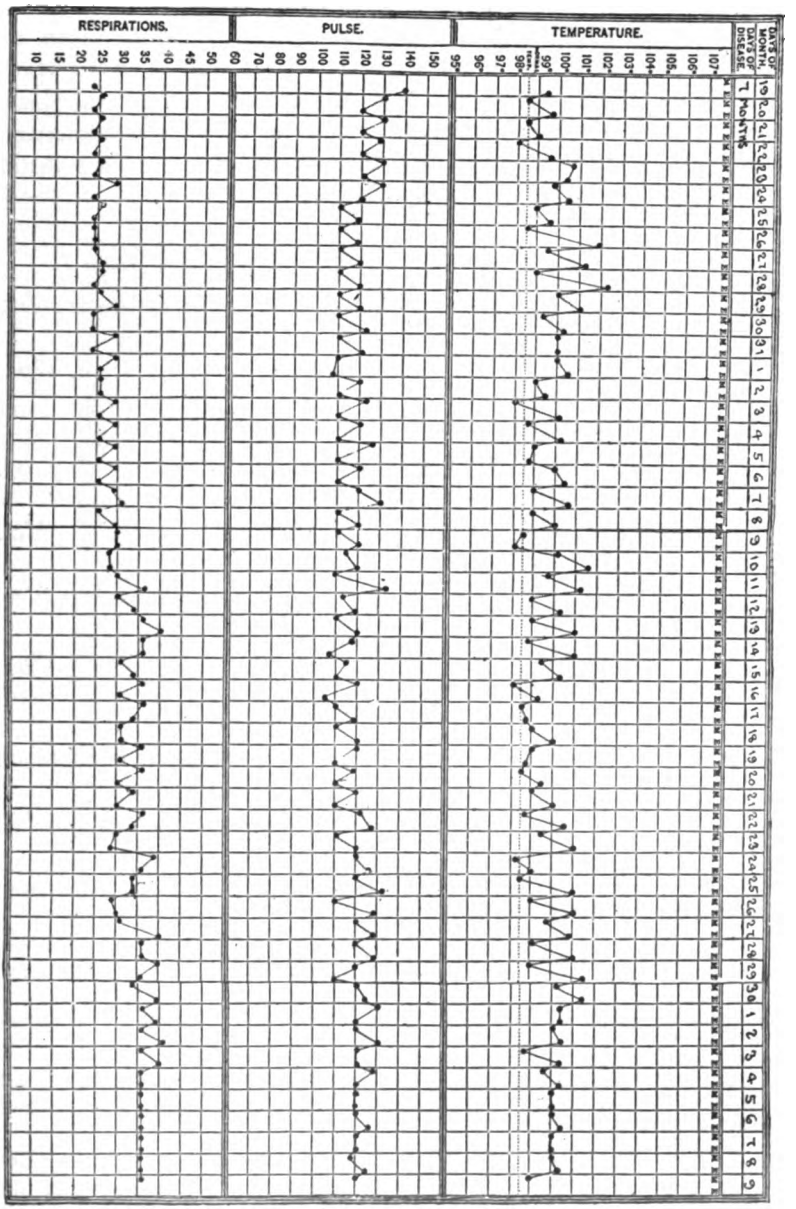

TEMPERATURE CHART OF A CASE OF PULMONARY OSTEOARTHROPATHY.

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and found to contain numerous influenza bacilli, free and in the leukocytes. A few staphylococci and rare streptococci.

The accompanying photographs are x-ray prints taken of the hands, feet and thorax. The x-rays show consolidation of the left lung, and a condition of the distal phalanges which correspond to the description which is given of pulmonary osteoarthropathy, that is, an enlargement of the entire distal phalanges, involving primarily the bone itself.

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Whitman. Pediatrics, 1899, Vol. VII., pp. 154-162; 14 cases; youngest, seventeen months, the only case under four years.

DISCUSSION.

DR. WEST.—For some time I have had under my care a case similar to Dr. Rotch's and I wish to take this opportunity of recording it.

This child first came under my care when she was three years and one month old. At this time she showed slight clubbing of her finger ends, but this was thought to be due to the habit of nail-biting, to which she was addicted, especially when excited. The father at forty-three years shows signs of premature age. The

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mother is thirty-three and well. The paternal grandmother died with tuberculosis. The paternal great-grandmother died at ninety-six after coughing for twenty years. About a month before the child began to cough she was under the immediate care of this old woman. There are two other children living, and six dead, all before they were a month old. The child has never been well; from her birth until her second year she would have severe crying and screaming spells, for which neither cause nor remedy could be found. These would occur at irregular and frequent intervals

HAND OF DR. WEST'S CASE OF PULMONARY OSTEOARTHROPATHY.

and gradually lessened toward the end of the second year. When eight months old she began to have a cough. This was dry, hoarse, and ringing and was worse and almost continuous after even slight exposure. After coughing this way for eighteen months she would expectorate some mucus after particularly hard coughing spells and would occasionally vomit from one to two tablespoonsful of thin, somewhat offensive mucus. When two years and nine months old she had a severe attack of measles; directly afterward the cough became harsh and hoarse and more continuous again for several weeks. At this time she was first seen by me. She was having the screaming spells again and a very severe cough. She was undersized, looked dull and stupid and her skin was sallow. She sat stooped forward with her back

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bowed. Her chest was symmetrical, rather narrow above and bulging below. No dullness. All over her chest and back, more so over her right back, were coarse bubbling râles. The heart and other organs were normal. A slight clubbing of the fingers was noticed; also, slight redness and roughness of the skin over the tips. As was mentioned, this was thought to be due to nail-biting, although the habit was not a pronounced one. She was now three years old. She was seen every two or three weeks for the next few months; her general condition improved very much; the lungs were considerably better; and the finger tips had not changed perceptibly. When she was four years old the fingers and toes were very much clubbed, the skin over the distal phalanges a bright pink and rough, and the nails short and broad. There was a slight stoop in her back, but no kyphosis. There was not, nor ever had been, any enlargement nor tenderness of any joints or bones. She has improved greatly in her general condition and disposition and the râles are smaller and fewer, the cough less frequent and severe. Four to six times a year she has a feverish attack lasting from twelve to forty-eight hours; outside of the high temperature and quickened respiration and circulation there are no symptoms. The râles disappear for the time and the breathing is harsh. The temperature drops quickly and she is soon herself again. In June of last year she had a severe attack of pleurisy with serous effusion. The effusion was absorbed and the disease had no perceptible effect on her condition either way. In the year past (the second under my care) the clubbing has increased slightly. (See photograph.)

With but brief intervals she has taken small doses of creosote regularly these two years; mostly by the mouth, and at times by inunction as recommended by Dr. Rachford.

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A REPORT OF EIGHT CASES OF PNEUMONIA IN INFANCY TREATED WITH ANTIPNEUMOCOCCIC SERUM.

BY JOHN LOVETT MORSE, A.M., M.D.,
Instructor in Diseases of Children, Harvard Medical School; Assistant Visiting Physician at the City Hospital and at the Infants' Hospital, Boston.

During the last dozen years many attempts have been made to produce a serum of therapeutic value in the treatment of pneumonia in man. The Klemperers were the first to produce immunity experimentally. They used filtered cultures and succeeded in obtaining a serum which protected other animals against fatal pneumococcus infection. They were able to demonstrate a protective body in the blood which they termed antipneumotoxin. Its therapeutic value was, however, very feeble. Its action was largely, though not entirely, antitoxic. Many others have since worked on the same lines with more or less unsatisfactory results. Other investigators have used sera obtained from men ill with, or convalescing from, pneumonia. Their results have been equally unsatisfactory.

Later investigators, among the first of whom were Washbourne, De Renzi and Pane, have, by the use of living cultures, produced sera which protected against virulent pneumococci and possessed a certain therapeutic value. These sera exert a direct destructive action on the bacteria but probably have little or no effect on the separable toxines of the pneumococcus, that is, they are antiinfectious rather than antitoxic in their action. Such sera are therefore usually known as antipneumococcus sera. The results obtained in the treatment of pneumonia in man with these sera have been on the whole discouraging, although some few observers have reported very favorable figures. Goldsborough about a year ago collected the cases treated with the various sera and published a summary in the Journal of the American Medical Association. The cause of the failure of the antipneumococcus sera to give more satisfactory therapeutic results is presumably the same as in the case of the other antibacterial sera, and

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that is, that while they are rich in the immunizing body, they are probably lacking in the end-body or complement. If this complement can be provided they will probably become more effective. Investigation along this line seems, therefore, most likely to yield results of value in the serum treatment of pneumonia.

Through the courtesy of the H. K. Mulford Co., who furnished me with the serum, I have been able to test the antipneumococcic serum in 8 cases of pneumonia in infants. All were patients at The Infants' Hospital, Boston. For lack of a better basis of classification, I have arranged them according to the dosage and total amount of serum used. The serum was injected every four hours, while the temperature was taken in the rectum every two hours. No antipyretic treatment was used except in 1 case which received a few baths. The other treatment was limited to regulation of the diet and stimulation when necessary. A careful physical examination was made in every case before the serum was begun, daily during its use, and for some days after it was omitted. The blood was examined before and after its use in only 3 cases.

CASE I.—Mary M., aged nine months, had had a cold for two weeks. She suddenly became much more sick and developed a high temperature on January 7th. She was admitted to The Infants' Hospital January 9th.

She was fairly developed and nourished. There was slight cyanosis. The alæ nasi moved with respiration. The heart was normal. There was slight dullness over the right lower lobe as far forward as the midaxillary line. At the root of the lobe, extending outward and downward about 4 cm., the respiration and voice sounds were bronchial. Over the rest of the dull area the respiration was diminished and bronchovesicular in character. The voice sounds were slightly increased and numerous medium moist râles were heard. The rest of the examination was negative except for slight evidences of rickets.

White blood corpuscles, 21,875. Urine: pale, turbid, acid, no albumin.

Five cc. of antipneumococcic serum were given every four hours.

January 10th. There were flatness with bronchial respiration. bronchial voice sounds and high-pitched medium moist râles over the right lower lobe as far forward as the anterior axillary line. There were numerous moist râles over the rest of the lobe in front.

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There were also a few medium moist râles in the left back. The color was no better.

January 11th. There was flatness with bronchial respiration over the same area as before but the respiration was diminished in intensity. There were a few high-pitched moist râles in the right lower back but none elsewhere.

The antipneumococcic serum was then omitted because the supply had failed. A total of 50 cc. was given.

January 12th. White corpuscles, 18,125.

January 13th. There were flatness with bronchial respiration, increased voice sounds and an occasional high-pitched moist râle over the right lower lobe.

January 14th. Paracentesis was performed for an acute purulent inflammation of the right middle ear.

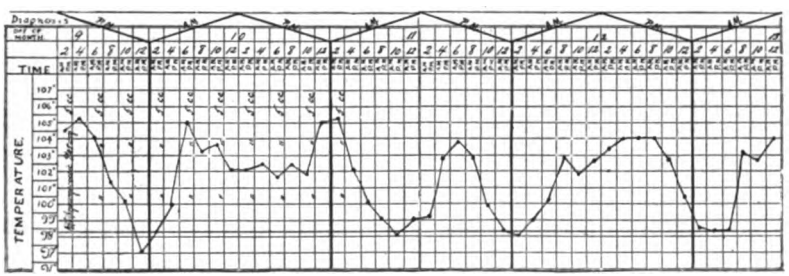

CHART I.

January 15th. The signs of solidification were less marked. The temperature fell to normal by crisis on that day and remained there.

January 17th. The lungs were clear. (See Chart I.)

This case came under observation so early and showed such definite signs that it seemed most suitable to test the value of the serum. The marked variations in the temperature shown in the chart are not unusual in the pneumonias of infancy. They were just as marked after the serum treatment was discontinued as before. The temperature both rose and fell immediately after the injections. The crisis occurred on the ninth day, rather later than is usual in infantile pneumonias. The solidification of the lungs increased after the serum treatment was begun. The rate of the pulse and respiration was unaffected. The conclusion seems justified, therefore, that the serum had no effect either on the course of the disease or its symptoms.

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CASE II.—Harry R., aged seventeen months, had had a slight diarrhea, and had passed but little urine during the three weeks since a vaccination. The history was, however, very indefinite. The physician who sent the baby to the hospital thought that the pneumonia began January 2d. He was admitted to The Infants' Hospital January 9th.

He was a large, fat baby. The fontanel was level. The alæ nasi moved with respiration. The heart was normal. There was marked dullness over the right upper lobe except in the lower part in front where there was tympany. There was bronchial respiration in the right front above the second rib inside the nipple line and above the fourth rib outside the nipple line. The voice sounds were bronchial over the same area. The respiration over the rest of the right upper lobe was bronchovesicular. The rest of the right lung and left lung were normal. The physical examination was otherwise negative, except for slight evidences of rickets. The general condition was good.

White corpuscles, 26,000.

Urine: high, acid, loaded with urates, albumin ½ per cent. or more. Sediment: urates, small round cells, a little normal blood, hyaline, fine granular and epithelial casts.

Five cc. of antipneumococcic serum every four hours was begun.

January 10th. The heart sounds were strong. The second sound at the pulmonic orifice was the louder. The color was not as good. The lips were dry and bleeding. The lungs showed in addition a very few râles in the right upper lobe. The urine was unchanged.

January 11th. Flatness with bronchial respiration and voice sounds had developed over the whole right upper lobe. No râles were heard. The rest of the chest was normal.

The antipneumococcic serum was omitted after 50 cc. had been given because of the failure of the supply.

January 12th. White corpuscles, 47,000.

January 13th. The lungs were unchanged. Paracentesis was done on both ears because of acute inflammation.

The temperature ranged normal after the 12th but never touched normal. The signs of solidification began to diminish on January 17th. The urine continued to show the same evidences of acute nephritis but was passed in fair amounts. The amount of urine fell off very decidedly on the 18th and very little

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was passed on the 19th. The urine on that day was thick and turbid and contained many small round cells, a very little normal blood, many hyaline, fine granular, brown granular and epithelial casts, and an occasional waxy cast. Marked symptoms of uremia developed during the 19th and death occurred in convulsions on the 20th. (See Chart II.)

The serum apparently had no effect whatever. The temperature was not influenced in any way. There was no reduction in the rate of the pulse or respiration. The physical signs increased while it was being used. The course of the disease was certainly not shortened. Inflammation developed in both middle

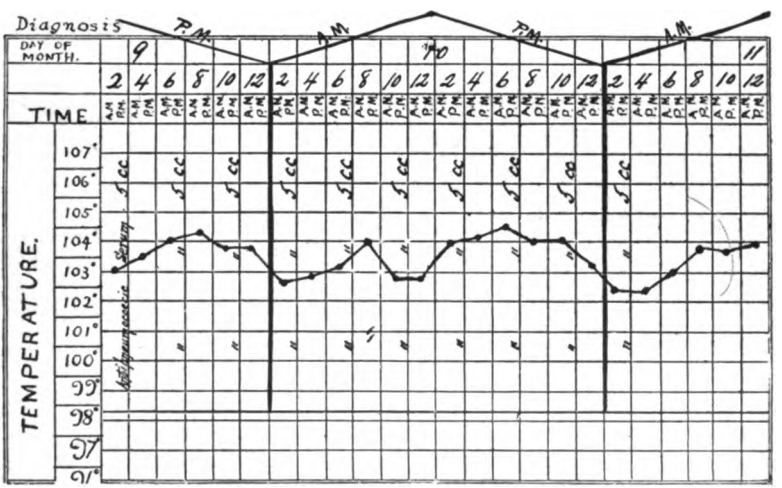

CHART II.

ears. Whether this inflammation was due to the pneumococcus or not is unknown as the pus was not examined. Presumably it was. Death was caused by the nephritis which was active at entrance before the serum was used. There is no reason to suppose that the serum aggravated the process in the kidneys.

CASE III.—David S., aged eight months, was admitted to The Infants' Hospital April 8th, on the fourth day of an acute illness.

He was well developed and fairly nourished. The color was good. The alæ nasi moved with respiration, which was grunting. There was slight retraction of the lower chest with inspiration. Resonance was impaired over the whole of both chests. There

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was bronchial respiration over the right lower lobe as far forward as the posterior axillary line and downward to the eighth rib. The voice sounds were increased in this area, and an occasional high-pitched râle was heard. The rest of the lungs was clear. The examination was otherwise negative except for slight evidences of rickets and slight enlargement of the spleen.

White count, 11,875.

The urine was high in color, highly acid and contained a trace of albumin. The sediment showed uric acid and an occasional fine granular and epithelial cast.

Five cc. of antipneumococcic serum were given every four hours.

April 10th. There was flatness in the area of bronchial res-

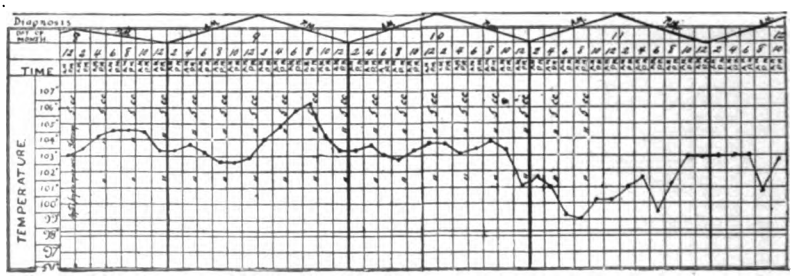

CHART III.

piration and crepitant and medium moist râles were heard over the rest of the right lower lobe.

April 11th. The signs were essentially the same although the râles were more numerous.

The antipneumococcic serum was omitted because of the fall in temperature. Ninety cc. had been given.

The temperature rose again, however, reaching 103° F. on the 12th. On the 13th it fell, that is the 9th day, by crisis and remained there. The lungs began to clear on the 12th and were practically normal on the 14th.

April 13th. White count, 16,016. (See Chart III.)

This case also seemed a favorable one to study as the physical signs were well-marked and the serum was begun on the fourth day. It had no apparent effect, however. The temperature both rose and fell immediately after the injections. In spite of repeated injections it remained elevated until the seventh day when there

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was a pseudo-crisis. The serum was then omitted. The real crisis appeared on the ninth day. The physical signs increased, if anything, while the serum was being used. The rate of the pulse and respiration was unaffected.

CASE IV.—Mary W., aged sixteen months, was admitted to The Infants' Hospital May 11th without any history.

She was fairly developed and nourished. The alæ nasi moved with respiration, which was grunting. There was flatness in the right front below the third rib extending backward to the post-axillary line. The respiration and voice sounds were bronchial in this area, and an occasional high-pitched moist râle was heard. The rest of the right lung was normal. There was slight dullness with bronchovesicular respiration over the left lower lobe, more marked at the angle of the scapula and in the axilla. The voice

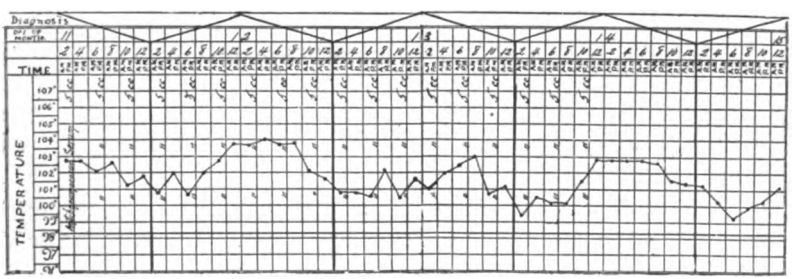

CHART IV.

sounds were somewhat increased and an occasional moist râle was heard. The examination was otherwise negative except for slight evidences of rickets.

Five cc. of antipneumococcic serum were given every four hours.

May 14th. There was no material change in the physical signs although the evidences of solidification were rather more marked on the left. There were râles throughout both lungs. The general condition was not as good.

The antipneumococcic serum was omitted after 90 cc. had been given, as it seemed to be doing no good.

The temperature continued to range irregularly elevated and an empyema developed on the right side. (See Chart IV.)

This case was undoubtedly one of bronchopneumonia and perhaps, therefore, hardly a fair one on which to test the serum, as

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bronchopneumonia is often caused by other organisms than the pneumococcus. The temperature showed considerable daily variations, which continued after the serum was omitted. The temperature both rose and fell immediately after its use. It had no effect on the rate of the pulse and respiration. The physical signs increased slightly while it was being used. An empyema developed later.

CASE V.—George G., aged eighteen months, had been a little out of sorts for two weeks but had shown no definite symptoms. He was taken suddenly sick April 11th and was admitted to The Infants' Hospital April 14th.

He was well developed and nourished, but pale. The alæ nasi moved with respiration. There was dullness in the right back above the middle of the scapula with slightly changed respiration and prolonged expiration. The voice sounds were not changed and no râles were heard. The spleen was considerably enlarged. The examination was otherwise negative except for moderate signs of rickets.

White corpuscles, 27,000.

Urine: high, acid, 1022, trace of albumin. Sediment: occasional small round cells, no casts.

April 17th. There was slight dullness with bronchial respiration over the right upper lobe, back and front. The voice sounds were slightly increased but no râles were heard.

The antipneumococcic serum was first given at this time, that is, on the sixth day of the disease. Five cc. were ordered every four hours.

April 18th. The signs were those of complete solidification of the right upper lobe.

April 20th. The temperature fell nearly to normal in the morning, but rose again in the afternoon.

April 21st. The right upper lobe showed no signs of resolution but the bases of both lungs were full of fine, moist râles. There was also edema of the feet and hands but more of the face. The urine was high, acid and contained a slight trace of albumin. The sediment showed an occasional small round cell and hyaline cast.

The antipneumococcic serum was omitted because the temperature was normal. One hundred and fifteen cc. in all were given.

April 23d. The edema of the feet and hands steadily in-

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creased and death occurred as the result of cardiac failure. (See Chart V.)

The temperature both rose and fell immediately after the administration of the serum. It practically reached normal on the ninth day of the disease, three days after the serum was begun. This fall, coming as it did on the ninth day, can hardly, however, be attributed to the serum. It came unusually late for an uncomplicated pneumonia in an infant. The serum had no effect on the rate of the pulse and respiration. The process in the lung became more intense while it was being used. Death followed as the result of cardiac failure due, in all probability, to toxemia. The serum was certainly of no utility in this case.

CASE VI.—Jennie F., aged twenty-two months, was taken suddenly ill June 9th with fever, cough and dyspnea. A diagnosis

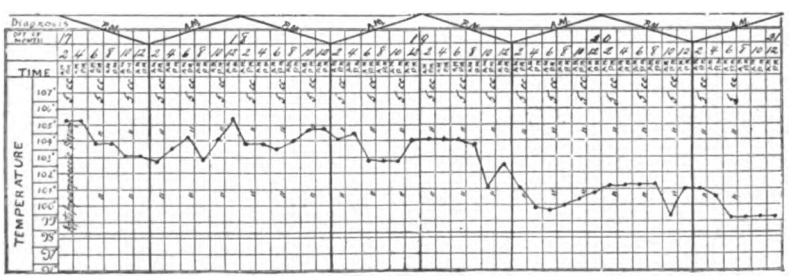

CHART V.

of bronchitis was made by some physician June 10th. The diagnosis of pneumonia was made by another physician June 17th and she was admitted to The Infants' Hospital the same day.

She was well developed and nourished and of good color. The alæ nasi moved with respiration. There was dullness with bronchovesicular respiration, increased voice sounds and occasional râles over the left upper lobe. The examination was otherwise negative except for slight evidences of rickets.

Treatment with the antipneumococcic serum was begun at once. Ten cc. were given every four hours.

June 18th. In the morning the left upper lobe showed all the signs of complete solidification. The general condition was somewhat better. The crisis occurred that afternoon and the temperature did not rise again.

The serum was omitted after the crisis, 70 cc. having been given.

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Resolution began June 19th, and the lungs were practically clear June 21st. (See Chart VI.)

The temperature both rose and fell immediately after the use of the serum. If we consider, as is probably true, that the pneumonia began on June 9th and that the diagnosis of bronchitis was wrong, then treatment was not begun until the seventh day of the disease and the crisis occurred on the eighth day. In this case, of course, it could not be attributed to the serum. If we assume that the diagnosis of bronchitis was correct and that the pneumonia did not begin until June 17th, then the crisis

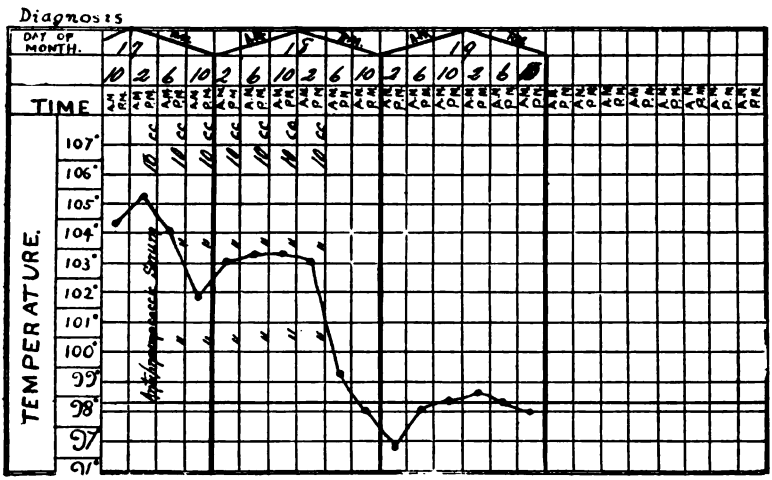

CHART VI.

occurred on the second day and might be attributed to the use of the serum. As, however, the signs of solidification were well marked at entrance, and there were no evidences of bronchitis elsewhere, the diagnosis of bronchitis was undoubtedly wrong. The crisis occurred on the eighth day, therefore, and was not due to the serum. The physical signs increased after the serum was begun. No effect on the rate of the pulse or respiration was noted.

CASE VII.—Frank T., aged twenty-four months, entered The Infants' Hospital April 28th with an indefinite history of a week's illness.

He was a well developed and fairly nourished negro. The alæ nasi moved with respiration, which was grunting. The right border of cardiac dullness was 3 cm. to the right of the median line. The heart sounds were louder to the left than to the right

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of the sternum. The left side moved less than the right. There was no bulging of the intercostal spaces. There was an area of flatness coinciding with the left lower lobe. There was slight dullness in Traube's space. There was loud bronchial respiration over the left lower lobe except at the extreme base where it was diminished. The voice sounds were increased; the fremitus was not determined. No râles were heard. The examination was otherwise negative except for slight signs of rickets.

Urine: high, acid, turbid, no albumin.

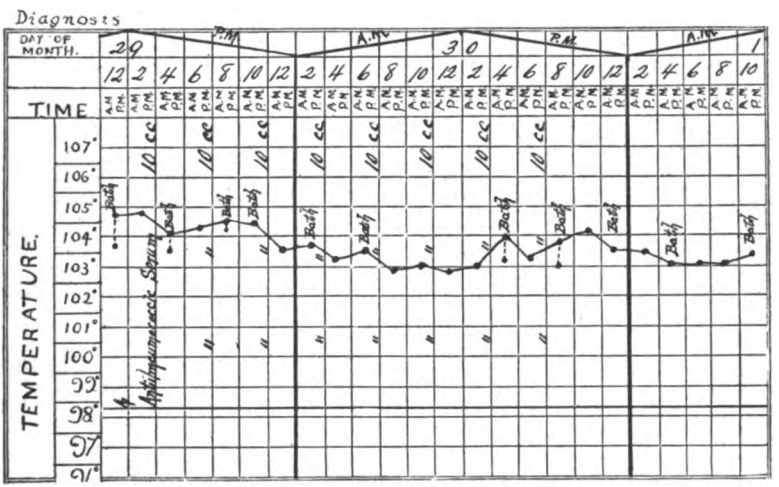

CHART VII.

April 29th. Treatment with antipneumococcic serum was begun, 10 cc. being given every four hours.

April 30th. The serum was omitted after 80 cc. had been given because the supply was exhausted.

May 1st. The heart was in normal position. There was dullness in Traube's space. There was flatness over the left lower lobe with bronchial respiration and voice sounds and an occasional high-pitched râle.

The temperature continued elevated. On May 3d the heart was somewhat displaced and there were the signs of a small amount of fluid in the left chest. Exploratory puncture was negative. The temperature continued elevated and the signs of fluid increased. On May 7th pus was obtained by puncture. The patient ultimately recovered. (See Chart VII.)

In this case the temperature neither rose nor fell after the use

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of the serum. The signs in the lungs did not change during its use. An empyema developed afterward. No effect on the rate of the pulse or respiration was noted.

CASE VIII.—Kaleel O., aged fourteen months, was admitted to The Infants' Hospital June 19th without history.

He was well developed and nourished and of good color. The alæ nasi moved with respiration, which was grunting. There was dullness over the whole right lower lobe with slightly modified vesicular respiration. The voice sounds were slightly, and the fremitus considerably, increased. There were no râles. The examination was otherwise negative except for slight evidences of rickets.

Urine: high, acid, no albumin.

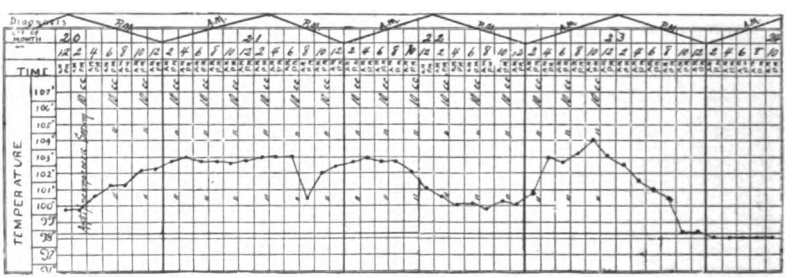

CHART VIII.

Although the temperature was low the antipneumococcic serum was begun on the 20th, 10 cc. being given every four hours.

June 22d. There was dullness over the whole right back with bronchovesicular respiration, increased voice sounds and fremitus. There was flatness over the right front with bronchovesicular respiration at the upper part. Elsewhere it was normal.

June 23d. The serum was omitted after 180 cc. had been given, because it was absorbed poorly. Both thighs were swollen, indurated and tender as the result of the injections.

The signs of solidification were somewhat more marked. The temperature fell to normal by crisis that night and did not rise again.

The signs of solidification quickly cleared up. The induration of the thighs was gone by June 27th. (See Chart VIII.)

This case shows the marked variations in temperature so often seen in the pneumonias of infancy. They were evidently not influenced by the injections of serum as the temperature both rose

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and fell after them. On account of the absence of history it was impossible to tell whether or not the serum shortened the course of the disease. The physical signs increased, however, while it was being used. This is the only case in which the serum was not well absorbed and caused any induration. This disappeared rapidly and did no harm. No effect on the rate of the pulse or respiration was noted.

In no case did the serum have any perceptible effect on the temperature. In 6 it both rose and fell immediately after the injection, while in 2 (2 and 7) the variations immediately after the injections were insignificant. In no case in which the time of the onset could be determined was the course of the disease shortened (1, 3, 5, 6, 7.) In fact, the crisis occurred later in these cases than is usual in the pneumonias of infants, coming on the eighth day in 1 (6) and on the ninth day in 3 (1, 3, 5). An empyema developed in the other (7) without there having been any fall in the temperature.

No effect on the rate of the pulse or respiration was noted in any case.

Several observers have noted that in adults the subjective symptoms were relieved for a time after each injection of serum. In infants it is, of course, extremely difficult to estimate variations in the degree of the subjective symptoms. Nothing was noticed in these cases, however, to justify the assumption that they were in any way modified by the serum.

The physical signs increased in 6 cases while the serum was being used (1, 2, 3, 4, 5, 8). In the others (6 and 7) in which the serum was not used until late in the disease there was no change.

Two (1 and 2) cases developed purulent inflammation of the middle ears, and 2 (4 and 7) developed empyema. Two (2 and 5) died; 1 (2) of nephritis, and 1 (5) of cardiac failure. In neither case was there anything to show that death was due to, or hastened by, the use of the serum. The serum was well absorbed in all the cases but 1 (8), in which it caused swelling, tenderness and induration about the seat of injection. These disappeared in a week. There were no rashes, joint symptoms, or late rises in temperature as the result of the injections.

The blood examinations were too few (1, 2 and 3) to be of any value, and the results were, moreover, not uniform. In an adult case on my service at the Boston City Hospital, in which examinations of the blood were made more often, there was usually a slight diminution in the number of leukocytes after the injection of the

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serum. Taken as a whole, there was a decided drop in the number of leukocytes after the serum was begun which persisted as long as it was continued. There was a slight rise again after it was omitted. (See Chart IX.)

CONCLUSIONS.—In these cases the antipneumococcic serum had no effect on the duration of the disease, the course of the tem-

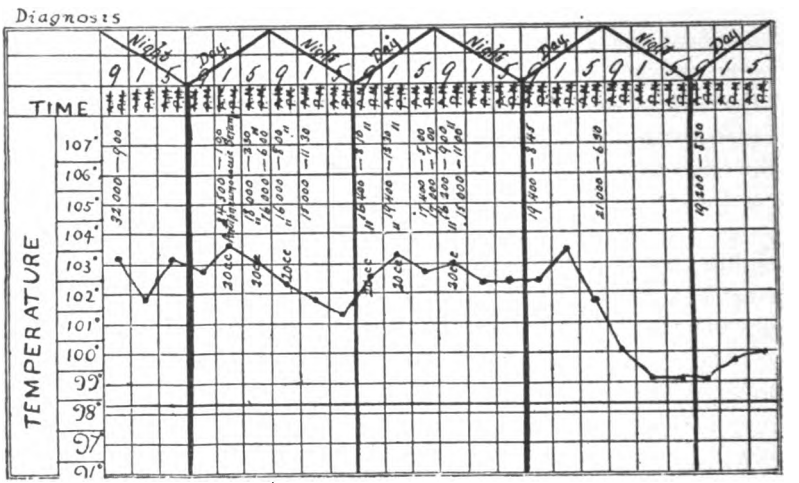

CHART IX.

perature, the rate of the pulse and respiration or the progress of the local condition. Complications occurred at least as frequently as is usual. Death occurred in an unusually large percentage. The serum, while it apparently did no good, certainly did no harm.

DISCUSSION.

DR. JENNINGS.—I had one experience in the use of the serum under conditions that would tend to show very clearly the effects of the serum upon the microorganisms in a case of pneumococcic meningitis. The serum was used within thirty-six hours after the onset of symptoms and repeated bacteriological examinations were made of the cerebrospinal fluid subsequent to the first injection. No influence whatever was made by the serum upon the clinical phenomena of the disease or upon the meningeal culture.

DR. SAUNDERS.—My experience with the serum has been just the same as that of the other gentleman. The last time I used it was, I think, about two years ago. I obtained no result whatever from the use of the serum and I abandoned it altogether.

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ABSCESS OF THE LUNG FOLLOWING ACUTE PNEUMONIA. A REPORT OF TWO CASES.

BY L. EMMETT HOLT, M.D.,
Professor of Diseases of Children, College of Physicians and Surgeons, (Columbia University), New York.

Abscesses of the lung of non-tuberculous origin are not uncommon in the autopsy room. They have been met with in my experience in about 7 per cent. of the autopsies upon infants and young children dying from pneumonia. Such abscesses are generally small, varying in size from a pea to a small marble; occasionally one as large as a walnut is seen. On one occasion I met with one which occupied nearly the entire lobe of the lung. Abscesses seen postmortem are seldom single, and frequently the affected part of the lung is fairly honey-combed with abscesses, some of them being very minute and others larger. These are usually seen in the lungs of children of low vitality, or have followed very intense inflammation, sometimes a very prolonged one.

Abscess of the lung as a clinical condition in young children is rare, the cases here reported being the first that have come under my personal observation.

Huber has twice reported to this Society (*Vide* Transactions American Pediatric Society, Vol. III., p. 235; Vol. V., p. 206) cases of pulmonary abscess successfully treated by operation. One of these was in a child thirteen months old, the other in one four years old.

CASE I.—ABSCESS IN THE LEFT UPPER LOBE OCCURRING TWO-AND-A-HALF MONTHS AFTER ACUTE PNEUMONIA; RECOVERY AFTER ASPIRATION.

J. P., male, eighteen months old, was admitted to the Babies' Hospital, December 30, 1902, with a history of having been taken ill with fever and rapid breathing two months before; more or less fever since, but not much cough. For one week was much worse, high fever, prostration, etc. Having grown too sick to be treated at a dispensary he was referred to the hospital.

The family history was negative as regards tuberculosis, and no other facts could be gleaned from the personal history, as the parents were Italians and not very intelligent. On admission the

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child was found well developed and well nourished with no important signs of disease except in his chest.

The examination of the chest showed coarse râles over the right lung; over the left lung there was dullness both in front and behind, and over the upper portion in front the percussion note was almost flat; loud, coarse friction sounds were everywhere heard. The respiratory murmur was everywhere bronchial in character, but over the upper half of the lung very feeble in intensity. Although there was no evident displacement of the heart, the flatness was so marked that an exploratory puncture was made in two places. No fluid was found. The case was therefore regarded as one of pleuropneumonia and its termination in empyema was anticipated. The usual routine hospital treatment for pneumonia was adopted. The child continued to look very sick; the cough was distressing; the respiration always very rapid, and occasionally there were attacks of slight cyanosis. The course of the temperature, the pulse and respiration record together with the leukocyte counts are indicated upon the chart.

For the first few days it will be seen that the temperature fluctuated irregularly between normal and 102.8° F. On the fifth day it took a sudden jump to 105° F. No marked changes in the physical signs occurred. The flatness over the upper lobe in front became gradually more and more pronounced, respiratory murmur was feeble, and friction sounds and coarse râles could be heard over nearly the whole chest.

On January 6th, although the physical signs remained practically unchanged, the leukocytes had gone up from 24,200 to 45,000. The presence of pus was now regarded as practically certain, and exploratory puncture again made, this time in the third intercostal space just outside the mammary line; a syringe full of thick creamy odorless pus was withdrawn.

Still under the impression that we were dealing with a sacculated empyema, preparations were made to remove the pus by incision. Chloroform was administered, but not to a degree to produce deep anesthesia. Desiring to drain the sac at the lowest possible point, a needle was inserted in the fourth intercostal space at the anterior axillary margin and pus found. An incision was then made at this point, but on opening the chest we came down upon the lung firmly bound by adhesions to the chest wall. The incision was extended upward and an opening made in the third intercostal space, but again the lung was met with as before.

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The child bore the operation and the anesthetic badly; his color was so bad and pulse so weak that any further operative measures at this time seemed inadvisable. Examination of the pus drawn by puncture showed the pneumococci in numbers and a few staphylococci.

On the following day the chest was aspirated in the third interspace at a point midway between the axillary line and sternum and 1½ ounces of thick creamy pus were removed.

From this time there was steady improvement in the child's general condition although the temperature continued to fluctuate widely. On the 9th of January exploratory puncture was made in two places at the upper part of the left chest in front but no pus could be found. A further steady improvement in the physical signs together with a gradual fall in the leukocytes convinced us that no further accumulation of pus had taken place. Further exploration, therefore, was not made although the temperature continued without apparent reason until January 18th. At this time the signs were dullness over the left upper lobe and feeble respiratory murmur; but the râles had gone; all the abnormal signs were much less distinct than at previous examinations and were gradually disappearing.

The boy remained in the hospital until March 15th. During the last month he gained three pounds in weight. The respiration continued somewhat rapid and he had a moderate cough evidently from the extensive pleuritic adhesions. On looking up the case subsequently it was learned that he had remained well for about a month after his discharge from the hospital, when he was taken severely ill with diphtheria and died in twenty-four hours.

CASE II.—ABSCESS IN THE LEFT LOWER LOBE TEN WEEKS AFTER ACUTE LOBAR PNEUMONIA. RECOVERY AFTER INCISION AND DRAINAGE.

H. P., male, two years, ten months; admitted to hospital, January 26, 1903, with signs pointing to a lobar pneumonia of the left lower lobe. The symptoms and physical signs were fairly typical. He had been acutely ill for five days. The family history was negative as far as tuberculosis of any hereditary disease was concerned. The patient had had bronchitis when an infant of six months, but had not been otherwise ill up to the commencement of the present attack. His temperature on admission was 104° F., pulse 140, repiration 56, leukocytes 39,500.

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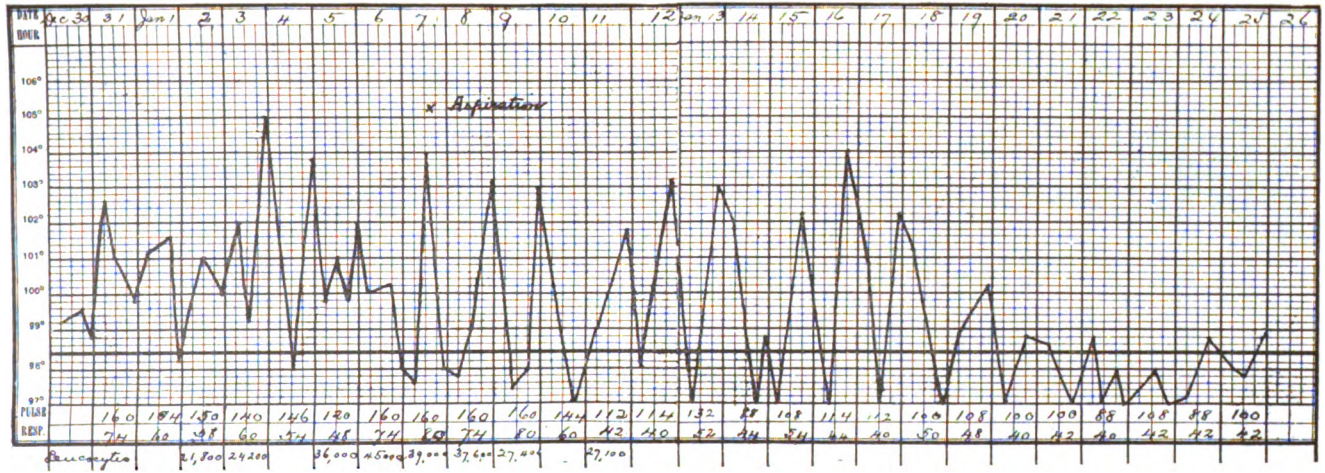
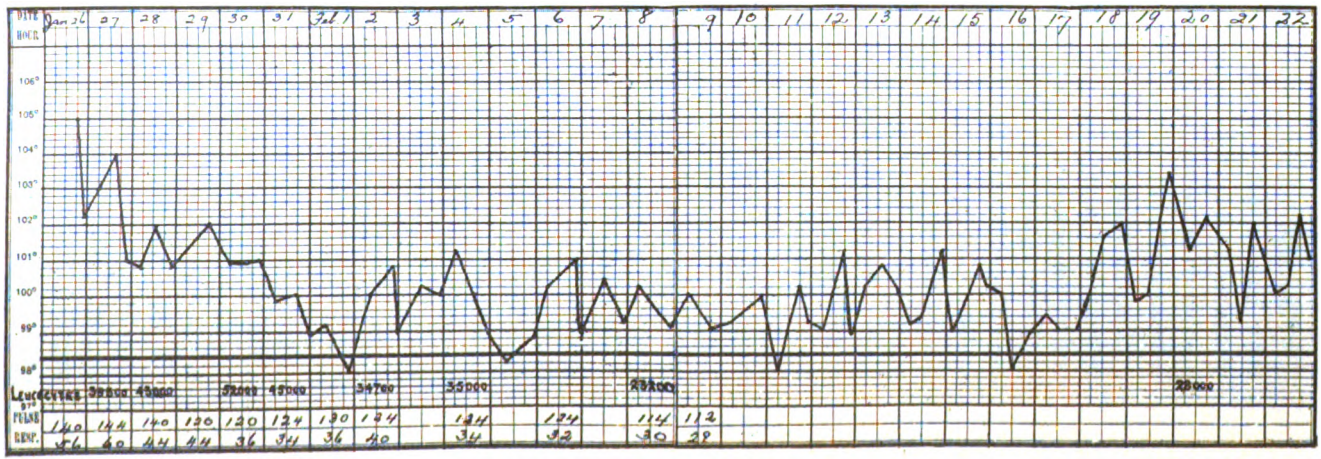


ABSCESS OF LUNG: CASE I.—CHART SHOWING TEMPERATURE, PULSE, RESPIRATION, LEUKOCYTE COUNTS AND DATE OF ASPIRATION.



ABSCESS OF LUNG: CASE II.—CHART SHOWING TEMPERATURE, PULSE, RESPIRATION, LEUKOCYTE COUNTS AND DATE OF OPERATION.

(See Continuation of Chart on next page.)

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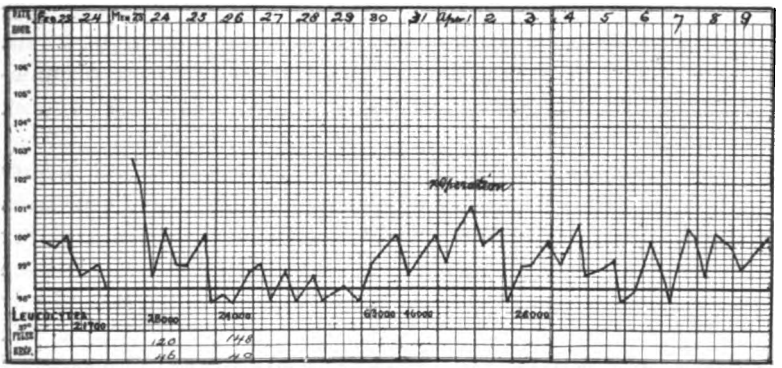

ABSCESS OF LUNG: CASE II.—CONTINUATION OF CHART.

By February 1st, the temperature had fallen gradually to normal, but remained so only a single day. The bronchial breathing disappeared but the dullness remained; and as the leukocyte count ranged from 35,000 to 50,000, it was expected that empyema would develop. The child was pale, had a poor appetite, looked badly, and lost weight. A slight, irregular temperature continued for the next five weeks. The physical signs remained practically unchanged; at no time was there marked flatness but always moderate dullness over the left lower lobe, together with feeble broncho-vesicular breathing, pleuritic friction sounds and coarse râles. We concluded it was probably a delayed resolution. On March 9th, he was removed from the hospital by his mother. The last leukocyte count on February 24th showed 21,700.

On March 23d, at the end of two weeks, he was re-admitted, having had in the interval an acute otitis which had ruptured, and the ear was discharging pus. From this time there was more temperature, prostration, and general evidence of illness. Examination of the chest showed no very great change in the physical signs. There was still present moderate dullness over the left lower lobe behind, with diminished voice and feeble breathing of a broncho-vesicular quality; a few friction sounds were heard; leukocytes 28,000. We felt tolerably sure now that empyema was developing, but greatly to our surprise the temperature soon fell to normal and remained there for four days. In spite of this there was no improvement in the symptoms and by March 30th, a rapid increase of the leukocytes to 63,000 convinced us that pus must be present. An exploratory puncture was made near the angle of the

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scapula with negative results. A second puncture at the same level, but a little nearer the spine, brought a syringe full of thick yellow pus. The examination of this showing only the pneumococcus, a diagnosis of sacculated empyema was made without hesitation.

On March 31st, the patient was given chloroform and further punctures made. It was not, however, until the fourth aspiration that pus was finally obtained, this being about one space higher than the site of the successful puncture previously. An incision was made at this point but we came down upon the lung firmly bound to the chest wall by old adhesions. A needle was inserted into the lung through the incision and pus found at a depth of about 1½ inches; there seemed now no possible doubt that we were dealing with an abscess of the lung. The firm fibrous adhesions which bound the lung to the chest wall obliterated the pleural cavity at this point, and made further operation comparatively easy. Following cautiously the line of the exploring needle an opening was slowly bored into the lung with a closed artery clamp, and a cavity reached at about the depth indicated, from which a gush of pus took place. About two ounces of thick creamy odorless pus was evacuated. The opening was enlarged with the finger and a rubber drainage tube inserted and the usual dressings of empyema applied.

The progress of the case from this time was uneventful. The discharge was quite abundant for four or five days; the drainage tube was gradually shortened, and after three weeks permanently removed. In another week the wound had healed completely.

The improvement in the child's general condition after the operation was immediate and striking. The cough disappeared, the boy gained rapidly in weight, and he was finally discharged from the hospital on May 25th; the only remaining signs being slight dullness and rather feeble breath sounds of a quality nearly normal. This child was heard from about four months after his discharge and reported to be well.

REMARKS.—Both of these cases followed acute pneumonia, the abscess being recognized about two and a half months after the attack of primary pneumonia. In Case II. the child was under observation almost continuously. In Case I., the history of the onset was fairly definite, and there can be little doubt as to the time when the process in the lung began. It is probable that a

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small abscess had been slowly forming for several weeks previous to its being recognized, and that it occurred in the center of an inflammatory area which had not resolved.

The bacteriological examinations of the pus are interesting. In both the pneumococcus was present; in one case it was associated with the staphylococcus; in neither case were tubercle bacilli found. These findings indicate that the abscess had been shut off from the bronchi by the products of inflammation, otherwise we would have had more evidence of mixed infection and doubtless foul pus. The presence of the pneumococcus gave additional weight to the supposition that we were dealing with an empyema. The fact that no pus was coughed up either before or after the operation also shows that no communication with the bronchus existed, although if the case had been allowed to go on it is probable that such would have been the result.

Perhaps the chief interest of cases like these is in the diagnosis. The literature of the subject shows that most of the cases of pulmonary abscess have been operated upon with the idea that a sacculated empyema was present, and indeed that was the first diagnosis made in both of my cases. In reviewing these histories critically, there are, however, a few points of differentiation which I believe may be of value in subsequent cases.

Percussion gives no clue to the nature of the disease. In both pulmonary abscess and sacculated empyema we have a more or less general dullness over the lobe, with flatness over a somewhat limited area. Again the voice and breathing sounds are practically identical in the two conditions. In both of my cases the presence of loud, coarse pleuritic friction sounds accompanying the signs on percussion and auscultation just mentioned, are I believe of much importance. Such signs could hardly exist if the accumulation of pus was in the pleural cavity. Another point of some value is the difficulty of finding the pus with the exploring needle, or finding it at one time and later not finding it even when a puncture was made in the same intercostal space. This should be regarded as evidence that the pus is deep-seated; not only must the needle be inserted in the same space but it must follow the same direction in order to find the pus. An abscess the size of a hen's egg, situated somewhat deeply in the lung might easily be missed although a half dozen punctures were made into the lobe which contained it.

A slight displacement of the heart would be probable in nearly

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all cases of empyema unless the amount of pus is very small. Of course, it would not be present in an abscess unless the amount of pus were large. This, therefore, could not be depended upon as an absolute point of differentiation.

A pulmonary abscess and a persistent or unresolved pneumonia may easily be confounded by physical signs. If an abscess exists it is often in the center of such a pneumonic area. Percussion gives little definite information; dullness often marked is present in both conditions. The circumscribed area of flatness would be found only when the abscess was large or superficially situated. Breathing and voice sounds over an abscess are feeble or absent, while over an unresolved pneumonia they are usually bronchial in character, though sometimes feeble in intensity. Coarse friction sounds are likely to be found in both conditions, often over the entire lobe if it were the seat of the antecedent acute pneumonia.

The physical signs of abscess, therefore, are not very characteristic. Additional help may be obtained from the other symptoms, especially the presence of continued fever and a high leukocytosis. The temperature curve in abscess is not characteristic, but some continuous fever is almost certain to be present. Fever is more constant and altogether higher than is usual in an unresolved pneumonia. A leukocytosis of 15,000 to 25,000 is often present in unresolved pneumonia, but a count of 40,000 or 50,000 and especially a rapid increase in the leukocytosis points strongly to the presence of pus.

From tuberculosis the diagnosis of abscess may be difficult and in some cases impossible without a prolonged period of observation. It should be remembered that abscess most frequently follows an attack of acute lobar pneumonia, that the patient may have been previously in the best of health and without any exposure to tuberculosis. Wasting is a less frequent symptom, tubercle bacilli are not found in the sputum and the pus found by puncture contains pneumococci, but no tubercle bacilli.

The treatment in the cases reported was comparatively simple. It was expected that the abscess would re-fill in the first case after aspiration. Incision is certainly to be preferred as a general method of treatment, provided the child's general condition is such as to allow the operation. Inasmuch as the pleural cavity is generally obliterated by dense adhesions, as in both these cases, the opening in the lung can be made with very little difficulty or risk.

14 WEST FIFTY-FIFTH STREET.

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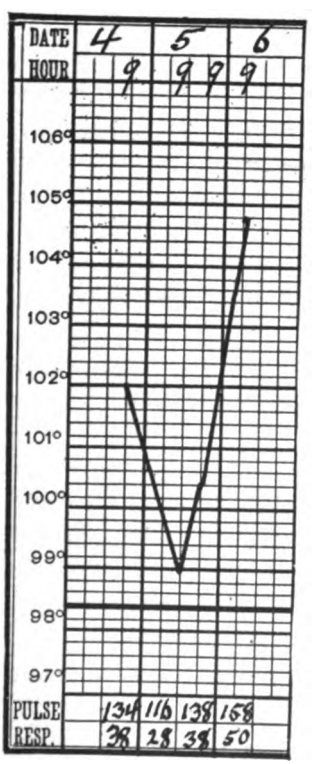
A CASE OF MULTIPLE ABSCESSES OF THE LUNG.

BY SAMUEL S. ADAMS, A.M., M.D.,
Washington, D. C.

As the subject of abscess of the lung is under discussion, I wish to report the following case:—

H. C., aged sixteen months, male, white, was first seen for me by Dr. Thomas S. D. Grasty on the afternoon of May 3, 1903, who ascertained that the child had been sick for several weeks with a cough and was then taking an expectorant mixture, together with an emulsion of cod-liver oil. He had not been seen by any physician during the preceding two weeks, during which time he had steadily grown worse.



TEMPERATURE CHART OF A CASE OF MULTIPLE ABSCESSES OF THE LUNG.

Dr. Grasty subjected him to a thorough physical examination and made the following report to me. Temperature (rectal) 103.5°; pulse 140; respiration from 60 to 80. He had an expression of distress and was very fretful. He had taken but little nourishment, and consequently had lost in weight. The various organs and systems, excepting the lungs, were apparently normal. Dr. Grasty decided that the patient had left pyothorax and that an operation was imperative. The next day I saw the child with Dr. Grasty, confirmed his diagnosis and had the patient taken at once to the Children's Hospital. He was admitted to my service and I again examined him at 4:20 P.M.

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His general appearance was about that of any child of his age; weight twenty-five pounds; dorsal decubitus; skin, muscular, osseous, glandular, digestive, circulatory, nervous and genito-urinary systems normal.

The chest showed breathing increased on right and greatly diminished on left side. Voice sounds feeble and distant on left side. Dullness on upper fourth and flatness over the rest of left chest. The patient was prepared for an exploratory puncture and I drew off with a small needle inserted below the angle of the scapula, a dram of bloody fluid, gelatinous matter and pus. This fluid was found to contain a large number of streptococci. He was transferred to the surgical ward for operation.

The next morning (May 5th) Dr. J. Ford Thompson resected a portion of the seventh rib and found the pleural surfaces thickened and adherent, but there was no pus. I entered the operating room just as he was applying the dressing, and one can imagine my amazement upon being informed that there was not a drop of pus in the pleural cavity at the site punctured by me the day before. The resident staff had seen me draw off the pus and the marked physical signs of fluid in the pleural cavity were present, nevertheless I was chagrined over what was regarded as a wrong diagnosis.

May 6th. The child did not react well after the operation, his temperature rose rapidly, convulsions supervened, and he died at 2:30 P.M.

The necropsy, performed by Dr. Mason, the resident physician, about an hour after death, was limited to the organs of the chest. Heart normal, right lung normal. Left lung in a state of delayed resolution; the whole being involved, and filled with large and small abscesses. I found that the needle of the syringe used by me on the 4th had punctured one of the larger abscesses.

The lung is here for your inspection, and you will see where I obtained the pus, as well as the multiple abscesses. The abscesses are not tuberculous. I had never before met with such a condition as this.

The thickened and adherent pleuræ and the pus in the multiple abscesses presented the physical signs usually found in pyothorax, hence the error in diagnosis.

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DISCUSSION.

DR. ROTCH.—It is not very unusual to puncture and get pus and then have the physician or surgeon not find it again for some time. We have quite a number of cases occurring in the hospital where, on the medical side, we aspirate and find pus, and send the case over to the surgical side to be operated upon and pus is not found. I have just had a case of empyema operated upon where the surgeon, when it was referred to him, sent word that he had tapped six times and had not gotten pus. He therefore allowed the patient to stay in the surgical ward for six weeks, and again tapped and found pus. The great point is to watch carefully, examine frequently and wait for it to come back.

DR. BLACKADER.—I think Dr. Adams is to be congratulated on finding pus. I have in the hospital gotten pus and have made the diagnosis of abscess of the lung, and I have myself aspirated when I felt sure a fleuroscopic examination showed abscess of the lung and I have not struck pus. Even when we make a diagnosis, we do not always strike pus.

DR. COTTON.—I think that lung abscess with pleurisy occurs more frequently than we are inclined to believe. A number of cases come to mind in which pus was found with the aspirating needle, but upon operation none was found in the pleural cavity. Such experiences as Dr. Adams' I have had twice.

Again the aspirating needle fails to find pus when the symptoms point to it and the physician feels sure he has pus. Later on there are symptoms of lung abscess, perhaps with breaking through into the bronchi with expectoration of pus, and then he thinks he had a lung abscess, when in reality it was a pleurisy with empyema, walled off by adhesions. This has been demonstrated several times in my practice, and has cleared up the mystery of these cases.

DR. ADAMS.—I made the diagnosis of empyema. I had just a short time before examined a child and made the diagnosis of empyema. The surgeon I called was not quite sure about it, and said: "Before I operate, I think we had better puncture." He put in the trocar and drew out the staff and pus fairly flew off. In this latter case there was a streptococcus infection, and no tubercles at all.

The specimen does not show well, as it has been in alcohol, but pus would come out through little openings in the lung by squeezing it.

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A NOTE ON ABDOMINAL AUSCULTATION.

BY HENRY L. K. SHAW, M.D.,
Albany, N. Y.

No mention is made of abdominal auscultation in any of our text-books on diseases of children. Inspection, palpation, and percussion of the abdomen are dwelt upon, but no importance seems to be placed on the results obtained with the stethoscope.

In infants and very young children the respiratory sounds are transmitted and can be distinctly heard all over the abdomen. All adventitious sounds, especially from the lower lobes of the lungs, and the lower and diaphragmatic portions of the pleura, are also transmitted. This fact is not generally recognized.

The respiratory sounds are synchronous with the respiration and are easily differentiated from intraintestinal sounds due to gas, fluid, etc.

My attention was first directed to this fact over a year ago in a case of what afterwards proved to be lobar pneumonia. A large, strong, nine months old baby was taken suddenly ill and went into a state of collapse. The abdomen was distended and there was no movement or passage of gas after a high enema and glycerin suppositories. The respiration was rapid with an expiratory grunt. The temperature was subnormal at first, but later rose to 103°F. Here was a condition apparently pointing to some abdominal disturbance. Four physicians were in consultation within twenty hours after the abrupt onset and agreed that the lesion was below the diaphragm, and that probably there was a general septic peritonitis. This conclusion was reached mainly on account of the results of abdominal auscultation, as a fine friction rub could be heard plainly all over the abdomen and below the diaphragm, posteriorly. Shortly after the consultation the baby had a large movement, and the abdominal symptoms improved. Crepitant râles were detected over the base of the left lung the next day, and the case developed into a typical attack

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of lobar pneumonia, followed by empyema. The râles could be distinctly heard over the abdomen for several days. This was a case of central lobar pneumonia at the base and it is interesting to note that the râles were heard over the abdomen before they could be detected over the chest.

Since this case I have listened over the abdomens in a large number of infants. Among these were 2 cases of lobar pneumonia at the base where the râles were transmitted as distinctly as in the first case described. In these cases several experienced diagnosticians auscultated the abdomen and corroborated my findings. Râles from acute bronchitis can be plainly heard over the abdomen. The more distended the abdomen the clearer are the transmitted sounds. The respiratory sounds are louder and more distinct in infants and the abdominal type of respiration is the rule. This, with the large size of the liver, may account for the transmission of the respiratory murmur.

Attention is directed to this point on account of its bearing on diagnosis. Disease, as manifested in infants, differs as do the physical signs from disease in later life, and all possible sources of error in the interpretation of physical signs should be borne in mind.

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SOME CASES OF INFANTILE NEPHRITIS.

BY F. M. FRY, B.A., M.D.,

Clinical Assistant, Royal Victoria Hospital, and Assistant Physician Montreal Foundling and Sick Baby Hospital,

AND CHARLES F. MARTIN, B.A., M.D.,

Assistant Physician, Royal Victoria Hospital, Montreal.

An examination of the literature of nephritis in infancy reveals it to be surprisingly deficient; indeed, there seems never to have been published, so far as we have been able to ascertain, any account whatsoever of a systematic examination of the urine in infants with a view to ascertaining both its general character, quality, abnormal constituents under varying conditions of health and disease, combined with a pathologic examination of those children who have subsequently died from various causes. Not indeed until J. L. Morse's article upon "Nephritis in Infants" appeared has any systematic work been brought out. This excellent work of Dr. Morse, which was published during the time our own investigations were in progress, shows that nephritis does occur perhaps more commonly than is supposed in infants, though his investigations do not seem to have been combined with pathologic examinations.

One of the difficulties encountered in such clinical investigations is the unsatisfactory means, hitherto devised, of gathering the urine. One of us (Dr. Fry) has used a method, however, which is eminently suitable for the purpose. For males of course the simple methods employed by others were of use here, though even in this particular one may fail where others succeed. It is essential to keep the bottle which served as the receptacle for the urine tightly bound down by means of a gauze bandage to the inner side of the infant's thigh, and the child should be made to lie upon the side until the specimen is obtained. For female infants, on the other hand, the older method, originally from Germany, of gathering the urine by means of a sponge, is certainly not satisfactory, at all events for the microscopic examination; nor can one

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say that the method of obtaining the urine by means of the napkin is any better. In either case the microscopic examination must suffer; and we believe, moreover, that this in itself may explain the hitherto unrecognized fact of how frequent are casts in the urine of infants. The use of a catheter is equally unsatisfactory for infants in the earliest weeks of life.

For our purpose we find *reflex stimulation* upon the bladder or upon the meatus would often promote the excretion of urine with some force and the stream could be made to flow directly into the bottle held for that purpose. This stimulation can be obtained by the application of cold above the pubis or by the mere manipulation of the orifice itself, and in almost every case the result was eminently satisfactory.

CLASSIFICATION OF INFANTS EXAMINED.—One hundred infants, under three months of age, were examined in all. They were selected from different institutions: Foundling, Maternity and Grey Nunnery. In this way we were enabled to obtain infants fed in various ways—by the bottle, by the breast, or otherwise —and this excluded to some extent the influence of dietetics upon the condition of the urine in various cases. Of the 100 infants, sixty-five were artificially-fed; thirty-five were breast-fed. With each infant there were at least three samples of urine taken on different days for examination; thus making in all over three hundred total specimens examined.

SPECIFIC GRAVITY.—The specific gravity, which was examined in the various cases, showed, as one would naturally expect, great variations due to the many conditions that exist in feeding, etc. The figures are, therefore, of but little value, though perhaps some may be of interest.

The average for the total number of samples was.....1 006.1
The average for the first 10 days.....1,006.4
The average from the 11th to 30th day..... ....1,005.1
The average from 2 to 3 months 1,007.5

It was certainly obvious that in the same infant increased specific gravity was associated with increased uric acid and great acidity in general. In one case where there was much uric acid present the specific gravity was 1,028; in many it was from 1,015 to 1,020; the lowest was 1,001; in other cases it was 1,003.

REACTION.—Sixty-four per cent. of all examinations showed acid urine, neutral 36 per cent.; there was none alkaline. It was

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observed that of the breast-fed children 78 per cent. were acid and 22 per cent. neutral; of the bottle-fed 91 per cent. acid and 9 per cent. neutral. It was noted, also, that the older the infants the more liable was the urine to be acid, that is, more than during the first ten days.

ALBUMIN.—In all instances the acetic acid and potassium ferro-cyanid tests were employed in addition to the cold nitric acid test.

Albumin was present in 19 out of 100 cases, varying from a trace to a very distinct ring by the cold nitric acid test. Of the 19 cases in which albumin was present 15 were bottle-fed children and 4 were breast-fed. Six of the 19 occurred in infants under ten days of age, 5 before the end of the first month and 8 during the second or third month.

CASTS.—Of these 19 cases with albumin, 17 had also casts, hyaline and granular mainly, a few epithelial casts but no blood casts. In 10 cases the albumin and casts were present in only one of the samples; in 7 other cases they were in two or more samples.

Of the 100 cases, however, 31 had casts, *i.e.*, 14 had casts without albumin. Of these 31, 22 were bottle-fed and 9 breast-fed.

URIC ACID.—This was in abundance in 26 cases. Of these there were albumin and casts in 14, and of the 12 remaining 10 had either albumin or casts, viz.: 9 had casts, 1 albumin. Casts were present in 23 cases.

It thus would seem that where uric acid was in abundance only a few, that is 3, escaped some evidence of nephritis or renal irritation. Of these 26, 19 died to our knowledge and autopsies were obtained on 7. In each of these there were uric acid infarcts and parenchymatous nephritis, and sections showed some marked degeneration of the convoluted tubules. In 3 of the cases there was in addition an acute interstitial nephritis. Apart from the 16 that died, 6 others had clinical evidence of nephritis, with marked edema about the face and limbs, as well as the signs in the urine.

With regard to the *relation of uric acid to nephritis* in infants, this was obviously increased in 26 cases. Of these 23 had casts, and of these 23, 16 died. Of the 16, autopsies were obtained in 9, 6 of which showed uric acid infarcts. Seven had uric acid in abundance and all of them had nephritis clinically; only 4 had no abundant uric acid with clinical nephritis. Whatever may be the relation of uric acid to nephritis one can say that the nephritis

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which occurs in infants is not alone associated with the toxic conditions incident to marasmus. That marasmus may often bring about a nephritis which, too, will be evident at autopsy, goes without saying; the fact, however, that identical conditions exist in the kidneys in children which were breast-fed and otherwise healthy, would indicate that the cause of the nephritis in many cases at all events is not due to the sole factor of marasmus. That it has been closely associated in some of these cases with the presence of marked increase of uric acid and that in many it is recovered from, may indicate a possible association between those two conditions, diseases which under favorable conditions are recovered from, without any serious permanent disturbance.

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A CASE OF DECAPSULATION OF BOTH KIDNEYS (EDEBOHLS' OPERATION) IN A BOY NINE AND ONE-HALF YEARS OLD.

BY THOMAS MORGAN ROTCH, M.D., AND H. W. CUSHING, M.D.,
Boston.

The boy entered the Children's Hospital, Boston, September 11, 1902. Family history negative. He was reported to have had measles three times, and to have been subject to tonsillitis. He was also reported to have had diphtheria and typhoid fever in the year previous, for which he was treated in the hospital. He was said never to have had rheumatism or scarlet fever. During his attack of typhoid in the previous year, the urine was reported to have been as follows: Color high; sp. gr. 1,022; reaction acid; sugar absent; albumin slightest possible trace.

Three or four days before his entrance to the hospital it was noticed that his abdomen began to be enlarged, and this was followed by swelling of the legs and then of the face. At this time there was noticed no change in the urine as to color and amount. He was said to have had dyspnea on exertion for some time previous to his entrance to the hospital. Had had no headache and no vomiting, but some dizziness at times. He was well developed and nourished; expression bright; skin clear, but of a pasty color; throat and tongue normal, but pale; teeth decayed; no enlargement of the lymph nodes. He had edema of the face and abdominal walls, and marked edema of the legs. The pupils were equal and reacted normally. Lungs were normal. Examination of the heart showed no increased cardiac area and clear sounds, excepting second sounds which were strongly accentuated. The abdomen was somewhat distended, but not tense and no ascites was detected. The blood showed: hemoglobin 50 per cent.; leukocytes 6,700.

The urine was smoky; reaction acid; sp. gr. 1,010; albumin

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⅛ to ¼ per cent.; bile and sugar absent; the sediment showed many hyaline and many granular casts with blood and renal cells adherent; a few fatty and epithelial casts; many round cells, often fatty; leukocytes free and in clumps; a few decolorized red corpuscles and a few squamous epithelial cells. Considerable free fat.

During the next three months the boy varied in his symptoms, sometimes growing better and sometimes worse. During this period he had at times considerable edema and a large amount of ascites for which he was tapped three times. The amount of urine also varied considerably from about 300 cc. to about 1,800 cc. The amount of albumin varied from ⅛ to ¼ per cent. During this time repeated examinations of the urine showed no marked difference from that found at entrance. The summary of the condition of the urine up to the time the child was transferred for operation is as follows: Color was at times pale and at times smoky; sp. gr. was about 1,010; the amount passed in twenty-four hours varied from 400 cc. to 800 cc.; at one time shortly after entrance a larger amount was passed, reaching 2,500 cc.; the amount of albumin was fairly constant from ⅛ to ⅓; the amount of urea excreted in twenty-four hours varied from five to ten grams; the sediment showed at all times very numerous casts both hyaline and granular, with renal cells and fat adherent; fatty and epithelial casts were usually present in smaller numbers, and at one time very highly refracting casts were noticed; fatty degenerated epithelium and free fat were constantly present in considerable quantities; renal cells were constantly numerous; the amount of blood in the sediment varied, at times there were many red corpuscles, free and adherent to the casts, and at others almost none.

The boy was transferred to the surgical department on December 28th, three months after entrance, and was placed under the care of Dr. H. W. Cushing. The following are the subsequent notes of the case:

His condition when coming under Dr. Cushing's care was that of a patient suffering from advanced parenchymatous nephritis. There was edema of the face, also of the back and of the external genitals, and marked edema of the lower extremities. The examination of the chest showed the heart's action somewhat irregular, rate 108, the cardiac dullness somewhat increased to the right. The lungs showed dullness and flatness in the lower lobes dorsally, especially at the bases, and medium and fine moist

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râles. The skin was very white. There was slight cyanosis of the extremities. The abdomen was distended, the circumference at the navel was 31 inches. The percussion was flat except in the umbilical region, and contained 4,220 cc. of fluid. There was occasional nausea and vomiting. The urine was diminishing in amount. From 1,000 cc. in twenty-four hours it had decreased to 480 cc. on December 29th. The specific gravity was rather low, 1,010; the amount of albumin varied from $\frac{1}{8}$ to $\frac{1}{3}$ per cent.; the urea varied from five to nine grams in the twenty-four hours; the sediment contained hyaline, fine granular, granular and fatty casts and fatty renal cells in abundance. The patient's general condition was growing steadily worse in spite of several months' careful treatment.

On January 1, 1903, Dr. Cushing decapsulated both kidneys. The duration of the operation was fifty minutes for the left side and thirty-five minutes for the right. Previous to this procedure 4,220 cc. of a slightly opalescent fluid, odorless, with neutral reaction, a specific gravity of 1,011 and a slight trace of albumin was removed from the peritoneal cavity.

The anesthetic was ether, preceded by nitrous-oxid. The technique of the operation was the usual one. The operation was more difficult than the average case on account of the edema of the dorsal tissues and the inability to deliver the kidney outside the incision of the skin. The decapsulation had to be done in the depth of the wound. The kidneys were enlarged; the fibrous capsules were not adherent; the denuded surface was matted with yellowish white areas alternating with injected ones; the cortex was quite friable. No especial cicatricial depressions of the surface were noted. The patient bore the operation well. The amount of ether inhaled was seven ounces. The pulse rate was 120 during the manipulation of the kidney, generally 100 to 110 at other times. The temperature fell to 94°F., three hours after the operation, and rose to 99°F., four hours later. For the next seven days it averaged 101°F.

The first act of micturition occurred five hours after the operation, the amount being 30 cc. During the first twenty-four hours the amount was 62 cc. It was slightly pale; very acid; urea 0.39 grams; albumin $^1/_7$ per cent.; the sediment contained numerous hyaline, finely granular and fatty casts, fatty renal cells and compound granule cells, leukocytes and epithelial casts; a few fibrinous casts and cholesterin crystals. There were more blood globules

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than in the previous specimens. The ratio of urine to the total amount of fluid ingested was 1 to 6.

There was distinct thirst after the operation, but the patient said he felt much more comfortable than before. The headache had disappeared, also the nausea. The edema also began to diminish at once.

The patient was temporarily relieved and lived until January 19th, on which day he died suddenly, with symptoms of acute pulmonary edema and cardiac exhaustion, eighteen days after his operation. The improvement noted immediately after the operation continued to January 8th, or 9th, and the condition seemed to indicate a favorable result up to that time. The patient was cheerful and quite comfortable. The edema had disappeared except in the legs and feet where it was much less.

The urine increased in amount from 85 cc. in twenty-four hours to 450, 1110, 1170 cc. The ratio of the urine to the total amount of liquids ingested had increased to 1:2; that is, from $\frac{1}{6}$ immediately after the operation to $\frac{1}{2}$..

On the seventh day the urea was 1.18 per cent., or 11.21 grams in the twenty-four hours. Albumin $^1/_7$ per cent. Sediment was still considerable, but less than at first. Casts were fewer, chiefly hyaline, granular, fatty and epithelial; an occasional fibrinous cast, blood globule and cholesterin crystal; a few renal cells and leukocytes were also seen.

From that time the clinical picture changed. The temperature rose to 105°F., and from that time until the end ranged between 104° and 100° F. The pulse averaged from 130 to 140 beats per minute. The operation wounds which had apparently united by primary union were partially strained open superficially on the thirteenth day by the returning edema. The respiration averaged 35. Nausea and vomiting recurred on the thirteenth day.

The urine diminished in amount from 600 cc. to 230, 290, 300, 350, and to 338 cc. on the fifth day preceding the death. The amount of albumin ranged from $^1/_7$ to $^1/_3$ per cent. The urea varied from 4.6 grams in the twenty-four hours to 3.6 grams. The sediment increased toward the end and its fatty elements increased, and there were numerous free oil globules.

The remarkable results produced by decapsulation of a seriously diseased kidney have attracted marked attention. Surgically, attention is not so much directed today to the possibility of the operation, or its technique, for both have been conclusively

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demonstrated, neither is it a matter of doubt that patients suffering with advanced nephritic disease have apparently recovered after this operation, but it has not yet been accurately determined what varieties of renal disease are benefited by this treatment, or what are the limitations of the operation. The surgeon has not yet the knowledge which enables him to decide in what cases decapsulation can be performed with success and in what ones it is contraindicated, also in what cases it will relieve even if the patient is not cured, and in what cases the result will be a fatal one.

The case just reported, although unsuccessful, may be of service in contributing data to assist in acquiring this desired knowledge and for this purpose is reported. The patient was in an advanced stage of the disease presenting distinct uremic symptoms. The function of the kidney was seriously impaired. There could be only one termination, and that in a comparatively short time. It was then an operation done as a last resort. The fatal result was unexpected after it was found that the patient did not succumb to the operation. It was hoped, as two weeks had elapsed before the condition became alarming, and as for a greater part of that time the patient had done well, that eventually he would recover. It was expected that the operation would be the severest strain. That he endured it so well was an encouraging feature.

It has been estimated that a period of from three to four weeks is required to fairly establish the new circulatory renal conditions which are essential for the permanent recovery. Hence in a patient whose renal tissue is so disorganized that his excretory power would be insufficient for that period, this operation would be contraindicated. It has been stated that the diminution of the daily amount of urea to, or less, than $\frac{1}{3}$ the normal amount indicates such deficient excretory power. In this case the rule holds good, although in the beginning the course of the disease seemed to indicate that it would be an exception.

The effect of the anesthetic was interesting. It was, although continued for nearly one and a half hours, in no way, as far as could be seen, deleterious. The character of the urine improved immediately after the operation.

The effect of the operation on the edema was also striking It was markedly diminished and continued so until three or four days before death, when it again began to reappear. This has been noted in other cases.

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DISCUSSION.

DR. EDEBOHLS.—I have recently published in the *Medical Record* (March 28, 1903) practically all that I have to say at present upon this subject. The matter, as Dr. Rotch has informed us, will come up in larger form at some of the meetings of the other sections and I may then find occasion to say something more. I agree with Dr. Rotch that the surgical treatment of chronic Bright's disease is not at all a settled matter, that it is still on trial, and that the final word will not be spoken for several years to come, until we know the ultimate results in the advanced cases which are at present being operated upon.

DR. RACHFORD.—How old was this case when operated upon; that is, how long had it had nephritis; and I should like to know whether the child died from the operation or from the nephritis?

DR. ROTCH.—The child had been ill for four months. It was apparently a perfectly healthy child, and I ought to have spoken of the result. So far as I can see what Dr. Edebohls has done, surgically, is to show that the operation can be done successfully.

I think my case was a case that would have died at any rate, from the character of the disease itself, but such cases must be investigated in the future.

The child collapsed at the end of two weeks and that comes within the period where the circulation is not restored in both kidneys. We cannot say that it was the effect of the anesthetic or the effect of the operation. It seemed to be the condition of the kidney itself.

DR. RACHFORD.—It appears to me that the future of this operation must be decided largely by operations upon adults, rather than upon children, since chronic nephritis is a much more common disease among adults than it is among children.

With children suffering from acute nephritis the tendency is to complete recovery, even though the disease may have been prolonged weeks or months. Chronic nephritis is, therefore, comparatively speaking, rather an uncommon disease in the child, while acute nephritis is very much more frequent at this period of life.

What Dr. Rotch has said concerning scarlatinal nephritis applies to all forms of nephritis whatever may be their cause. In all of these the tendency is, if the patient lives a certain length of time, to ultimate recovery, although a small percentage may run into chronic nephritis.

In these cases therefore it is difficult to say whether the operation brought about the recovery, or whether they would have recovered without the operation. In cases of chronic nephritis, however, which you recognize as an incurable disease we are safe in crediting the operation with the recovery, because we know they could not have recovered without the operation.

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DR. JENNINGS.—I might note one observation which bears upon what Dr. Rotch has said in regard to the possible functional disturbances as a cause for those symptoms. The case was one of chronic renal insufficiency and chronic uremia with severe paroxysmal pain and great tenderness over the left kidney. The case came to operation and it was thought there might be renal calculus. The urinary findings were those of a chronic interstitial nephritis. The kidney did not present, macroscopically, any marked pathological change, at least it did not present the appearances of an advanced chronic interstitial nephritis. The capsule was split and partially peeled back and the pelvis explored for stone, etc. The woman made a rather remarkable recovery. There was restoration of renal function, entire relief of pain and an improvement in her general condition so that she appears to be now, in two or three months, a pretty well woman. Here is a case presenting, macroscopically, no apparent pathological change in the kidney, but with the urinary findings, of a chronic interstitial nephritis.

DR. ROTCH.—I should like to say a few words in reply to Dr. Rachford. In former times I agreed with Dr. Rachford that chronic nephritis in children was very rare, indeed so rare that when I saw my first case I looked upon it as something extremely unusual. Of later years, however, I believe that it has been proved not to be so and we have chronic nephritis in children just as we do in adults. We have been having quite a number of chronic nephritic cases in the last few years under our care in Boston at the Children's Hospital, and we have seen them die of chronic nephritis just as adults do, and I believe that it should not be conceded that it is so rare in children to have chronic nephritis.

To be sure it is comparatively rare just as chronic diseases in children are in comparison to chronic diseases in adults; but that does not alter the question that it occurs, and if it occurs we should bring forth all the information we can to determine when these cases should be operated upon. A certain class of these cases may be saved by Dr. Edebohls' operation. Of course. it is the wonderful recuperative power of the child which makes chronic disease, or death from chronic disease, so much more rare in childhood than in adult life. It seems to me that there is going to be a very broad field for Dr. Edebohls' operation among children. We must decide whether these cases in children should be operated upon. Since chronic nephritis occurs in children they may die from it. It will be a great advance in treatment when we have learned to pick out the class of cases for operation. Cases following scarlet fever though likely to get well, when they become chronic, still at times die and they are perhaps the ones whose lives might be saved by Dr. Edebohls' operation.

DR. RACHFORD.—I did not mean to say that there was no field for this operation in infancy and childhood. I did, however,

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say that it would be more difficult to prove the value of Edebohls' operation, from operations in infancy and childhood than in the adult, because it was more difficult in the child to decide when these cases were chronic and incurable.

If they are operated on, for example, in six months of the time when the albuminuria began, there is at the time of the operation a chance of their getting well under medical treatment, for this reason it would be impossible to state positively that a surgical operation had cured these cases. I do not mean to say, however, that cases of this kind are not to have the benefit of this operation, but only insist that the true value of this operation must be largely decided by operations upon chronic and incurable cases, such as are commonly found in the adult.

DR. COTTON.—I have nothing to say more than the information that it has been my good or bad fortune to have 4 cases in the past year of chronic nephritis in children under four years of age. There were three deaths; two autopsies. One child still under treatment is two and a half years old; and, although of course every year my practice does not bring that number by any means, still I have begun to think that it is not so rare a disease in early life as we used to consider it.

DR. ADAMS.—I want to understand the pathological condition of the urine of the infant. I should like to know whether any one can recall an advanced case, such cases as we know go on from bad to worse, in which the child recovered, where there were found the evidences of structural changes of interstitial nephritis, because that would have a very great bearing upon the point of contention, as one of the speakers admits that these cases invariably go on to destruction. Has he ever seen a case where the evidence of interstitial nephritis was so marked in which the child recovered?

DR. ROTCH.—I do not believe that this must be decided by results in the adult. Even if the operation turns out to be a success in adults, I believe we still ought to decide whether it ought to be done in children by a separate study of children. In one we have the developed and in the other the undeveloped organ. I think it is a large field which we have before us. It is always more difficult to make a diagnosis or a prognosis in children than in adults, no matter what disease they may have. We should not go to the adult to decide whether certain treatment is good for the child any more than we should go from the child to the adult.

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CHRONIC DIFFUSE NEPHRITIS IN A FOUR-YEAR-OLD BOY WITH A RUDIMENTARY KIDNEY.

BY A. C. COTTON, M.D.,
Chicago, Ill.

RECORDED CASES.—The rarity of solitary kidney is evident from the limited number of cases found on autopsy. Holt mentions but 1 case of single kidney in a thousand consecutive autopsies, in which twenty renal malformations were found. Ziegler says: "Congenital atrophy of one kidney is more common than entire absence. In well marked cases the atrophied kidney appears as a thin plate of fibrous tissue 2 to 5 cm. in length and 1½ to 3 cm. broad, with few or no traces of tubules or glomeruli, and is supplied by renal vessels normal in position but abnormally small."

G. B. Fowler states that in 11,978 autopsies made at the four largest London hospitals only three instances of marked atrophy were observed and that in 1894 not more than 70 or 80 cases had been reported. From an analysis of a large number of autopsies Morris concludes that single kidney is found only once in 2,400 bodies. Ballowitz in Virchow's Archives August 5, 1895, reports as a result of his researches 213 cases of absence of kidney.

ETIOLOGY.—As to the etiology of this interesting condition Ziegler states: "The causes which determine the non-development of one of the kidneys are unknown. We can only say that for some reason the outgrowth from the primitive ureter out of which the kidney is fashioned has been hindered or altogether suppressed. In some cases, however, traces of inflammation, in the form of cellular infiltration and fibrous hyperplasia, are discovered in the rudimentary organ. We are thus led to infer that intrauterine inflammation of the kidney is possible and may lead to arrest of its development."

The American Text-Book of Pathology suggests that renal hypoplasia may be due to faulty development, to congenital smallness of renal artery, to intrauterine inflammatory changes or to congenital stricture of the ureter. Although it has been stated that we have twice as much renal parenchyma as is necessary to carry on elimination, the statement may well be questioned from the fact that reported cases of hypoplasia of a kidney show compen-

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satory hypertrophy of its fellow. Our autopsy shows no exception to this rule, the single kidney showing marked enlargement.

It is not strange that half the cases of absence of one kidney show a tendency to disease in the remaining organ. That the gravity of nephritis, either acute or chronic, should be manifold in these patients is self evident.

The following report embodies the history of a case of a boy of four years who had a rudimentary kidney.

HISTORY OF CASE.—*Family History.*—Father living and well, has four brothers and one sister all healthy. Mother living and well, has three brothers and four sisters, all healthy. Patient has three sisters, two healthy, one with infantile cerebral paralysis. One brother died of cholera infantum and one sister died ten days after birth.

Personal History.—Was a normal baby. First severe illness was in October, 1900, when he had an attack of bronchopneumonia, lasting twelve weeks. Since then has never been strong, catching cold readily. During these colds the mother noticed the urine decreased in quantity, was often passed involuntarily and was of an ammoniacal odor. In January, of 1902, edema of ankles and lower lids first appeared, disappearing and recurring at varying intervals under treatment. The child had measles in April, 1902, after which there was first observed the distention of the abdomen which still persists.

Physical Examination.—Child is fairly nourished. Eyelids are closed by edema. Abdomen is distended and fluctuating. Ankles edematous. All other points negative. Slight cough without physical signs.

Urine.—Examination of twenty-four hours' specimen of urine showed the total amount to be 480 cc.; specific gravity, 1,012; urea, 1.6 per cent.; urea, total, 7.68 gm.; total solids, 12.8 gm.; albumin present; hyaline and granular casts present.

Treatment and Record.—The patient was put to bed, placed on liquid diet and freely supplied with water and lemonade. Morning and evening he was given hot baths, followed by thirty minute hot packs which usually caused abundant diaphoresis. Citrate of potash was administered in small doses and later normal salt enemata were employed for diuretic effect.

This treatment, varied to suit the symptoms, was continued until the middle of October. Under it the patient would improve for a time, the edema and ascites would almost disappear and he was

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allowed to sit up and even walk about the ward. Then, suddenly, edema would reappear, the pulse and respiration would become accelerated with a slight rise of temperature. During this period the patient urinated from eight to twelve or more times each day, always in small amounts. The total amount varied from 300 to 900 cc. for twenty-four hours and the specific gravity from 1,007 to 1,013. Albumin was always present in considerable amount; urea percentage was low and the total urea was small.

The temperature ranged from 98° to 100° F., occasionally reaching 100.5° or even 101°. The pulse rate varied from 96 to 150, usually ranging between 110 and 130. The pulse was often weak and sometimes irregular.

During the latter part of August an eczematous rash appeared on the head and face, to a lesser degree upon the body and limbs and persisted for several weeks. On September 15th the patient had improved so much that he was given light diet in place of the liquids. His medicine consisted of carbonate of ammonia in 2 grain doses; diuretin in 5 grain doses four times a day, and magnesia sulphate in 2 dram doses each morning.

From October 10th to 20th the patient was troubled with vomiting and pulse was often irregular and weak. He was again put on liquid diet and given diuretin in 5 grain doses, with strychnia, $^1/_{120}$ gr., four times daily. On October 22d calomel, in doses of $^1/_{10}$ grain every two hours, was ordered, and magnesia citrate, 2 ounces, given every morning. The result of the treatment was quite a severe salivation which began on October 25th, with a rising temperature, which reached 102°F., and gradually subsided by the 29th. The pulse ranged from 120 to 160, was weak and often irregular. The teeth were slightly loosened, tongue and lips much swollen; face and eyelids markedly edematous. The submaxillary and parotid glands were enlarged and tender; the breath was foul and lips excoriated.

About December 15th the patient developed some impetigo contagiosa which proved unusually obstinate. The general symptoms again improved and he was given a soft diet. Sweats were ordered once instead of twice daily. Until February, 1903, nothing occurred; the patient was better and worse at intervals, with a general downward tendency.

On February 10th a morning specimen of urine showed a very large amount of albumin, many hyaline and granular, and a few waxy casts. The temperature began to show a subnormal ten-

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dency, frequently as low as 97° F., by rectum, with a diminution in the amount of the urine. Urination was frequently involuntary so that the total amount could not be accurately determined. February 14th, while the ward was being fumigated, the patient was transferred to a room on the sixth floor of the hospital in which, during the extremely cold weather that followed, a proper temperature could not be maintained. The sweats were necessarily discontinued and the child grew rapidly worse. On the 18th of February he urinated only three times, a total of 180 cc. The urine was loaded with albumin. The specific gravity was 1,010, with a few hyaline and many granular and fatty casts. The following day he passed urine twice, a total of 112 cc., and on the 20th there was almost complete anuria. During these three days the patient was much prostrated with tendency to semicoma. Physical examination showed considerable fluid in the abdomen and lower part of the thorax. Auscultation revealed large numbers of moist râles throughout the lungs. Respiration was rapid (40 to 60 per minute), labored and apparently painful. The breath was heavy with a urinous odor. The temperature was subnormal and pulse weak and irregular, ranging from 100 to 156. Muscular twitchings and tremor began on the 18th, but no general convulsion occurred. On the 20th the patient was returned to the children's ward and given a hot bath with no apparent effect. The pulse became extremely rapid; the respiration shallow and the patient died at 2:30 P.M., two and a half hours after the bath.

During this patient's nine months residence in the hospital his urine was examined 100 times; nearly forty of these examinations being twenty-four hour specimens in which the total amount, specific gravity, percentage of urea, total urea, total solids and albumin, were carefully estimated. The table of findings is reduced to general averages as follows:—

	Total amount of Urine.	Specific Gravity.	Urea, per cent.	Urea total.	Total solids.
Average	623	1,010.8	.759	4.87	14.29
Highest	1,200	1,018	1.6	18.00	32.48
Lowest	300	1,006.	.5	1.80	7.00

AUTOPSY RECORD BY PETER BASSOE, M.D.

Necropsy of Charles Byron Clark, age four years, date of death, February 2, 1903. Date of autopsy, February 2, 1903; clinical diagnosis, chronic nephritis.

External Examination.—Child well nourished; skin pale;

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slight edema of legs and chest; rigor mortis slight; small abrasions on right index finger and thumb; length of body one meter.

Internal Examination.—Subcutaneous fat 1 cm. thick; peritoneal cavity contains one liter of clear serous fluid; peritoneum smooth and presents no abnormal adhesions; liver extends 3 cm. below costal arch in right mammary line. There is a large Meckel's diverticulum.

Thorax.—Each pleural cavity contains 250 cc. of straw colored fluid. On the right side are a few frail bands of adhesions uniting the parietal pleura with that covering the lung. The left side is free from adhesions. The pericardium contains 2 ounces of clear fluid. The thymus gland:—In its position is a thick layer of fat in the centre of which is a firm tissue. The thyroid gland:—Size proportionate to age: glistening, pale grayish red on section. Larynx: —Mucous membrane smooth and pale. Trachea:—Mucous membrane smooth and pale; trachea-bronchial lymph nodes a little enlarged and pale red in color.

Lungs.—Left, weight 120 gms.; bluish gray and smooth externally; floats in water. Upper lobe crepitates freely; on section reddish, but contains very little blood; tissue tough, permeated by heavy bands of fibrous tissue. Lower lobe a little darker on cut surface, otherwise the same in appearance as upper. Right, weight 130 gms.; floats on water; fissure between upper and middle lobes partly obliterated. Upper and lower lobes very firm and crepitate very feebly. Middle lobe crepitates freely. On section the right resembles the left lung closely, except that in the lower lobe a little mucus can be expressed from the smaller bronchi.

Heart.—Weight 130 gms. Contains small amount of fluid blood. Tricuspid opening admits three fingers and mitral opening 2. All the valves are smooth. Left ventricle is 6 cm. long; ventricular wall 1 cm. thick. Right ventricle is 5 cm. long; wall is 2 mm. thick. Circumference of aortic opening is 44 mm.; of pulmonary opening 45 mm. Foramen ovale is closed. Myocardium is pale.

Mouth and pharynx normal on inspection, esophagus smooth.

Abdomen.—Spleen, weight 50 gms.; externally light reddish in color; cuts with increased resistance; trabeculæ prominent on section.

Stomach.—Size proportionate to age, contains some sour smelling milk curds; mucous membrane smooth and of normal appearance.

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Small Intestine.—Lining is smooth throughout. 80 cm. from ileocecal valve is a Meckel's diverticulum 6 cm. in length and as thick as the index finger; no constriction at base; at free end are two shallow pouches containing a small amount of mucus. Appendix:—9 cm. in length, normal in appearance.

Large Intestine.—Solitary follicles prominent, especially in upper part; mucous membrane smooth throughout.

Pancreas.—Weight 30 gms.; cut surface pale; markings are distinct.

Liver.—Weight 550 gms.; external surface smooth, pale; cuts with slightly increased resistance; cut surface pale; small amount blood; lobular markings very distinct. Gall bladder contains thick greenish bile; walls thicker than normal.

Adrenals show no changes; both of usual size.

Right Kidney.—Weight 65 gms.; length 7 cm., width 4 cm., thickness 3.5 cm. The perirenal fat is considerable in amount. The capsule strips easily but leaves a very irregular surface with prominent stellate veins and many minute depressions and elevations. Cut surface is a mottled light grey. Cortex is pale, the distinction between cortex and medulla very slight. Cortical markings are very distinct. Mucous membrane of the pelvis is smooth.

Left Kidney.—Rudimentary, consists of a small triangular whitish body with base upward. It measures 1 cm. in width and 1½ cm. in length. (See cut.) From this body an artery and a vein can be traced to the aorta and ascending vena cava respectively.

Right ureter normal in size and position. Left ureter, length 13 cm.; inserted into bladder in normal position; terminates above in the rudimentary left kidney described. The ureter possesses no demonstrable lumen.

Bladder, prostate, testicles and seminal vesicles normal. The epiphyseal cartilage at the lower end of the femur is 1½ mm. thick; line of ossification perfectly regular.

Anatomical Diagnosis.—(1) Extreme hypoplasia of left kidney. (2) Chronic diffuse nephritis (right kidney). (3) Hydroperitoneum. (4) Hydrothorax. (5) Hydropericardium. (6) Edema of legs and thoracic wall. (7) Hypertrophy of left ventricle. (8) Slight bronchitis. (9) Fatty liver. (10) Persistent Meckel's diverticulum. (11) Chronic interstitial pneu-

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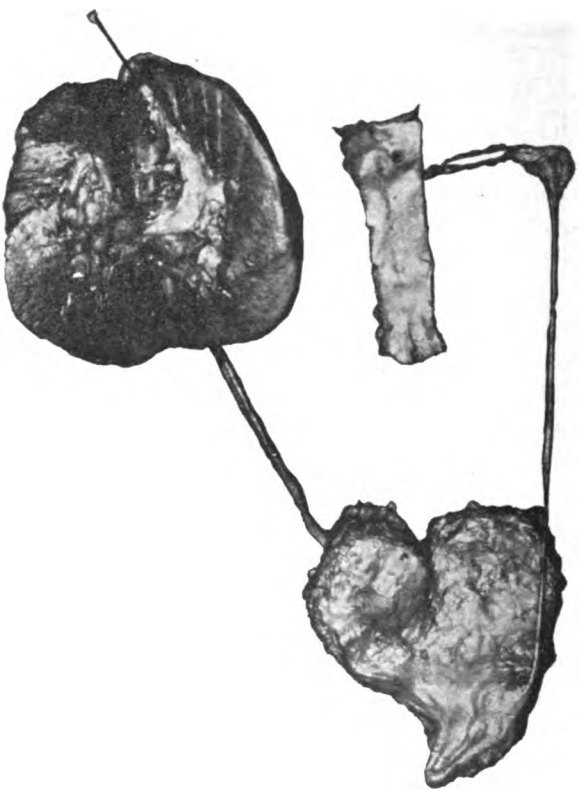
monia. (12) Hyperplasia of solitary follicles of ascending colon. (13) Localized fibrous pleuritis.

Histologic Findings.—Only those concerning the kidneys will be given.

Right Kidney.—A great many of the tubules are dilated and contain hyaline masses. Other tubules contain leukocytes and



RUDIMENTARY LEFT KIDNEY.

epithelial debris. In the cortex and more so in the pyramids are found accumulations of round cells many of which are polymorphonuclear leukocytes. In the cortex are hyaline fibrous glomeruli. The condition is one of acute inflammation engrafted on a chronic diffuse nephritis.

Left Kidney.—The rudimentary left kidney was cut in serial sections, made horizontally, if we imagine the kidney in its position in the erect body. Each group of ten sections was placed in

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a separate numbered bottle and two or three sections of each stained by hematoxylon and eosin. The numbers used in the following description apply to these fifty-five groups, each roughly representing $^1/_{55}$ of the length of the kidney.

Nos. 1 to 4.—Sections are made up of a dense fibrous tissue with scant elongated nuclei, and considerable evidence of hyaline degeneration. In No. 4 is a straight row of rounded nuclei which suggests a tubular structure which becomes plain in No. 5. In No. 6 and No. 7 the hyaline degeneration becomes more pronounced. In No. 8 we see a cross section of a tubule the diameter of which is a little more than half of that of a normal glomerulus. It is lined by a single layer of low epithelium and contains homogeneous material which takes a pink stain, and may be said to constitute a hyaline cast. In No. 9 two structures are seen which appear to be imperfect glomeruli. In No. 10 and 11 a couple of additional tubules are seen in longitudinal section.

Beginning with No. 7 a rather large space has been noticed in the interior of all sections. In No. 12 it is plainly seen to be lined by a single layer of flat epithelium. It is narrow but extends over more than the central half of the section. The latter measures 10x2 mm. This space contains a homogeneous faintly stained material.

In the succeeding sections several new tubules are encountered some of them lined by a tall cylindrical epithelium. In No. 15 several such tubules of the convoluted type are seen in longitudinal section, but there are no glomeruli. In No. 17 is a glomerulus-like structure which is partly calcified. Near it are several fairly well-formed tubules of convoluted type. From No. 25 downward the tubular structures diminish in number and the central space becomes smaller and indistinct. The sections mainly consist of hyaline fibrous tissue, with a few tubules surrounded by a more cellular connective tissue and a number of thickened blood-vessels.

In No. 32 the tubules again become more numerous. In No. 35 is a small collection of mononuclear round cells around a group of tubules. In No. 48 the interior cavity and its epithelial lining become plainer and persist to the last section (55). In all sections the blood-vessels are quite numerous, except in the hyaline interior portion, and their walls are thickened. The cavity mentioned as occupying the interior of most of the sections may represent the pelvis or possibly merely a cyst. At no point were tubules seen to empty into it.

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A CASE OF INFANTILE MYXEDEMA.

BY GEORGE N. ACKER, M.D.,
Washington, D. C.

M. V., aged two years and ten months, female, white, entered my service at the Children's Hospital, Washington, D. C., February 4, 1903, with the following history:—

Her mother and father are living and in good health. She is the only child, born after a normal labor. She was breast-fed for sixteen months. About the fourth month she cut four upper incisors and about the eighth month the four lower incisors. She had no more teeth until the eighteenth month, when the anterior molars and canines appeared. She began to sit up at the sixth month and commenced to talk at the ninth month. When eighteen months old she could walk short distances, and make sentences of a few words. She was a fine looking, healthy child. At that time she had an attack of diphtheria, for the treatment of which antitoxin was employed. Her general appearance began to change after recovery from the disease, but it was six months before any great change was noticed. It was then observed that the face was swollen, the skin thickened, the upper teeth decayed, and bronchial wheezing was marked. For two weeks before she entered the hospital she did not recognize her mother.

She had ceased to grow in height, but her weight greatly increased during the past year.

CONDITION ON EXAMINATION.—"The skin is very firm and thickened, the arms and legs appear hypertrophied. The chest is round and the abdomen is distended. There are masses of subcutaneous fat about the clavicles. The anterior fontanel is slightly open. The nose is flat. The hair is straight and dry. The tongue is large, broad, flabby, and appears too large for the mouth. The appetite is fair and the bowels costive. The thyroid gland cannot be felt. She has a slight cough, and examination of the lungs shows a few mucous râles. The heart is normal; pulse 120 per minute. The child is dull and listless. There is evident lack of intelligence. She does not talk, nor in any way express her wants. She does not walk. Sensation is good. There is involuntary micturition."

TREATMENT.—She was ordered ½ grain of thyroid extract

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three times daily, and in four days this was increased to a grain. This was continued for two weeks when 1½ grains were given. On these small doses, which were given on account of high pulse rate and bronchitis, the child made a marked improvement and lost weight rapidly.

February 20th it was noted that she was brighter, less irritable and played with her toys. From this date until the middle of March she took 3 grains of the extract three times daily. She had a severe attack of influenza from the middle of March to the middle of April, with a discharge from the left ear, and during this illness the thyroid extract was reduced to 1 grain three times a day. The patient's condition was so greatly changed for the better that it was recorded on April 8th that her appearance was nearly normal. The face had lost its bloated appearance; the expression was brighter and intelligence much increased.

When with her mother she spoke a few words. For some weeks she passed urine voluntarily. Her weight was 23 pounds and 6 ounces, and her height 30 inches. During July and August the child was taken to the seashore. The medicine was continued in 1 grain doses, three times daily, until January 21, 1904, when, on account of an attack of influenza with high fever, it was discontinued for a week. In the early part of March, 1904, the patient had the chickenpox from which she made a good recovery.

March 28, 1904. Since the child has been under treatment she has taken on the average 1 grain of thyroid extract three times a day, and there has been a wonderful change in her condition. She appears to be a bright, healthy child, plays with the other children in the ward and enjoys life. Her skin is soft and pliable. She walks about and talks, but not as well as a child of her age should do. The digestive and genitourinary systems are in a good condition. The fontanel is closed. Her face has an intelligent expression, and the nose has lost its flat appearance. Her weight is 35½ pounds and height 35 inches. She has gained about 7 inches since her admission to the hospital.

DISCUSSION.

DR. KOPLIK.—The Society may be interested in the subsequent history of a case I reported a number of years ago. Among the first cases I published was a child nineteen months of age when brought under my observation. It was of the myxede-

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matous type. I continued my treatment, although the girl never became so bright as other children of her age, until she was about seven years old. She was then brought to me with the query as to whether the medicine should be continued. The child was fairly bright and went to school. I discontinued the thyroids and it was not more than a month before the symptoms of myxedema reappeared, but in a more aggravated form than when I first saw the case. There were great masses of fat over the clavicles and the appearance was that of a very marked cretin. The child was very stupid and, of course, had to return to thyroids again. This proves that these little unfortunates are not cured permanently by thyroids. They must be kept under constant observation.

As to the administration of the thyroid, it is astonishing to see what large quantities children can stand, if the thyroid preparation is absolutely pure. Gregor, in a recent publication, has attempted to find out how much thyroid extract a baby could tolerate. He administered large doses, as much as 150 grains a day to a child three years of age, without any ill effect, and he concluded that, if there is a rise in temperature after the administration of thyroid tablets or extract, we should consider that our preparation is not absolutely pure. In the early days of the administration of thyroid extract there was a marked toxemia due to impurities and toxins in the thyroid extracts which at that time were frequently made from raw glands, partly decomposed.

DR. ROTCH.—In Boston we stop our treatment when we get a rise in temperature. The quantity of the thryroid to be administered is an important thing. If the preparations are not pure we should not give them.

DR. COTTON.—I should like to know whether it is the experience of others, in using the thyroid treatment in a myxedematous condition, that they have observed a greater tendency to ordinary infections, such as acute coryza or influenza? In a number of cases that I have handled I have found that a prolonged use of the thyroid improved the general physical condition but increased susceptibility to incurring infections; so that the child, who had been absolutely free from any disturbances whatever, except this one of development, became a very delicate and sensitive child. I reported some of these cases several years ago.

DR. BLACKADER.—Is the thyroid gland palpable here?

DR. ACKER.—No; it is not. A great improvement has taken place in this child. She is mentally brighter; the tongue is smaller and the hair is less dry; she walks now by herself, and has lost the puffy condition of the skin; the bridge of the nose is much better; and the skin and muscles have a normal feel.

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THE ETIOLOGY OF RACHITIS.

BY ROWLAND G. FREEMAN, M.D.,

Lecturer on Pediatrics, University and Bellevue Hospital Medical School; Attending Physician Foundling Hospital; Nursery and Child's Hospital, New York.

In the following paper I wish to summarize briefly some experimental work which has been carried out to determine the cause of rickets, and to refer to some collective observations made by me on three small groups of infants from hospitals and tenements in New York, and finally to discuss the conclusions that may be drawn from these.

Although the disease has been recognized and studied for 250 years we are still very much in the dark as to its etiology. It was first described by Whistler in 1645 and was first called to general attention by Glisson's description of the disease in 1650. In the nineteenth century a more careful study of the disease was made with very extensive experimental work on animals.

In considering the etiology of rachitis we must agree that it is not a disease of the bones alone, but a general disease affecting the bones, ligaments, muscles, nervous system and mucous membranes and that the earliest symptoms of the disease, the sweating of the forehead and the restlessness, as indicated by a bald spot on the back of the head, are often unaccompanied by evident bone lesions. Although rachitis is said to occur between the sixth month and second year it is my experience that it is often fairly developed before the sixth month. That is, before that time the signs of craniotabes, the bald spot on the back of the head, the sweating of the forehead, and the rosary may be observed.

The theory of Parrot that rachitis is always of syphilitic origin is not advocated at present. A deficient supply of oxygen seems to be an important cause of rickets or, as we may say, climate is an important etiological factor. Rickets being essentially a disease of temperate and cold climates and almost unknown in hot climates, where people do not shut themselves up in close rooms, naturally those who suffer most in our temperate climates are those who come from hot climates, as the negroes and Italians. There is no doubt but that rickets is much more common in this country in Italians and negroes than in foreigners from northern climates,

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and our worst cases of rickets are seen in these people and especially in the negroes. The frequency of rachitis in certain temperate and cold climates is shown in the following observations:—

In Russia Ionkowsky states that 95 per cent. of the infants show slight lesions of rickets, while 35 per cent. show severe lesions. In Norway Johannessen considers that 32 per cent. of the children of his clinic have rickets and that the cases are seen most frequently in April to June, and least frequently in November and December. In Boston, Morse found that 80 per cent. of the infants at his dispensary class showed rickets. In Greece, Algiers, Peru, Mexico, and India and other countries with warm climates this disease is said to be seldom or never seen.

Rickets is, moreover, a disease of the cities and is much less frequent where people live less closely. Indeed, it has been suggested that rickets is a carbonic acid poisoning, but this theory has lacked support. That cold climate and the accompanying bad air of closed and close rooms is alone a sufficient cause of rickets I do not believe, but undoubtedly unsuitable food will produce rickets more rapidly when the child receives an insufficient supply of oxygen. It has thus been stated by several observers (Kassowitz, Wallach in Munich, Mey in Riga and Bystrow in St. Petersburg) that rickets occurs more frequently in winter than summer. Although race and climate may predispose to rachitis the active cause seems to exist in the diet.

The determination of the exact cause of rickets would seem to be rendered more simple by the fact that this disease may be produced in most of the lower animals, but in examining such series of experiments we find few consistent or conclusive results. When it was known that the bone lesions of rickets showed a deficiency of lime salts the theory was at once advanced that the deficiency of the salts in the food was the real active cause of the disease. It was indeed found by Chossat that by withholding lime salts from the food a softening of the bones resulted but a typical rachitic condition was not produced. Moreover, cow's milk contains more lime salts than mother's milk and again rickets is very common in places where there is much lime in the water. Another modification of this theory is, that the trouble is in the failure of the system to absorb the lime salts (Henoch and Baginsky), but observations on the elimination of lime salts in the urine of the rachitic and normal subjects showed no difference. It has been further held by Pommer that, although sufficient lime

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may be fed and absorbed, it may not be deposited in the neoplastic substance on account of diminished alkalinity of the blood, but it has been shown by Stoeltzner that the alkalinity of the blood is not diminished in rickets.

Again the lesions of rickets have been attributed to the presence of lactic acid in the system or, as is held by Monti, an excess of lactic acid in the stomach with a lessened amount of hydrochloric acid. This lactic acid theory may appeal to us because of the fact that many of the foods which seem to produce a severe type of rickets contain starch which may produce lactic acid in the stomach and, also, on account of the large content of lactic acid and lactic acid bacteria in some market milk. Extensive experiments on animals by many observers, consisting in the injection of lactic acid have failed almost always to produce rickets. Spellman injected a rabbit for a month and a half with 10 cc. of a 1-200 solution of lactic acid without producing any bone lesions. On the other hand rickets occurs in animals on a diet which does not lead to the development of lactic acid. Guerin fed young animals on meat instead of mother's milk and they developed rachitis. In the London Zoological Gardens more than twenty litters of lion cubs, which had always been fed on meat, were lost from the development of rickets. At last one litter with advanced rickets was seen by Bland Sutton who advised for them milk, powdered bone and cod-liver oil with meat. They were left with the same environment but in three months they showed no signs of rickets. These animals were thus dying from rickets on a diet of proteid, only of a sort rather difficult of digestion, and were cured by a diet of more digestible proteid with fat. And this leads us to inquire whether the absence of fat in the food may not be an important factor in the production of rickets.

Clinical experience has convinced many of us that much of the severe rickets is produced by feeding with proprietary foods and condensed milk, both of which are very poor in fat. Thus, while mother's milk contains usually 3 to 4 per cent. of fat, and two to three times as much fat as proteid, we find in the ordinary condensed milk less fat than proteid, and in the pulverized proprietary foods from one-fourth to one-fourteenth as much fat as proteid.

Experiments were undertaken by Dr. Herter, of New York, to produce rickets in pigs by fat starvation. One pig was fed fifty-one weeks on a limited quantity of Walker-Gordon skim milk

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containing $^1/_{40}$ per cent. fat, while sow's milk usually contains 8 to 10 per cent. fat, so that the pig received only $^1/_{800}$ to $^1/_{400}$ the normal amount of fat. During the fifty-one weeks the pig gained 16 pounds. It, however, became constipated, weak and its skin became dry and it was drowsy. It was moribund when killed. Another pig was fed twenty weeks on an unlimited quantity of the same food. It also developed muscular weakness and drowsiness. A third pig was fed fifty-six weeks with skim milk and carbohydrates. This pig developed the muscular weakness and had occasional diarrhea. Other pigs were also used. As a result of the experiments and autopsies on the pigs Dr. Herter concluded that fat starvation in pigs does not cause rickets. No rickety lesions in the bones of these pigs being evident. The most peculiar discovery at the autopsies was the presence of mucoid degeneration, which replaced the fat of the adipose tissue. This mucoid degeneration did not, however, occur in the animals that received an excess of carbohydrates.

It thus seems fortunate that these canned foods for babies contain usually an excess of carbohydrates, since by analogy this fact may have saved the babies fed on them from a mucoid degeneration of their adipose tissue, although subjecting them to fat starvation.

Delcourt, in 1899, held that the lesions of rickets could be produced in animals by the action of phosphate of potash and that he had produced them by the administration of phosphate of potash to dogs. Spellman repeated his experiments. He gave to seven rabbits for a month daily doses of 2 to 5 grams of phosphate of potash. To a young pig he gave 700 grams of phosphate of potash in twenty days. To another rabbit one month old he gave for several days subcutaneous injections of 5 to 50 cc. of a 5 per cent. solution of phosphate of potash. All the animals were killed and showed no macroscopic or microscopic evidence of rachitis.

Spellman, although having failed in obtaining positive results, believed the lesion due to some irritation and thought it most likely that such irritant had its origin in the gastrointestinal tract. In order to determine whether intestinal poisons would cause the lesions of rickets he made repeated injections into twenty rabbits and one cat of extracts of the diarrheal stools of rachitic infants, and in only one of these animals, a rabbit, did he obtain evidence of rachitis. Experiments in feeding animals on the colon bacilli, and others in injecting their toxins, also failed to produce rachitic

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lesions. The inoculation of cultures of the staphylococcus and in other experiments of their toxins gave negative results. The toxins of the bacillus pyocyaneus gave a like negative result. Morpurgo, however, states that by injecting into young rats a pathogenic diplococcus he obtained typical rachitis. Efforts made by Spellman to isolate a specific etiological organism from the intestine of rachitic infants have also proved unproductive. The theory that intestinal toxemia is the cause of rickets is opposed by Kassowitz who calls attention to the fact that intestinal toxemias are a summer disorder, rickets a disease of winter. In like manner the bone of rachitic infants has been found to contain the same organisms as the bone of nonrachitic infants. The inoculation into healthy animals of bones removed from rachitic infants have in 5 cases given negative results. Notwithstanding his 126 failures and one positive result Spellman is not discouraged and is still pursuing his investigations.

In considering all this experimental work one must remember that one positive result is of the greatest value; while negative results, although of great value for reference in future experiments, should not produce much influence on our opinions, and especially we should not apply to children any deductions from experiments on animals which gave negative results. This statement is well supported by experiments of Spellman in which he subjected various animals to the combination of the conditions we believe to produce rickets in babies: poor air, poor light, defective forms of food, etc., so that in all ten died from gastroenteric disorders in from one to four months, but not one showed any sign of rachitis.

If then we review the positive results of these experiments we must reject those of Delcourt in which phosphate of potash was administered, since they lack confirmation, although repeated by Spellman. Morpurgo obtained positive results in rats from the injection of a pathogenic diplococcus. There is, however, no confirmation of this observation.

Spellman in one experiment obtained rachitis from the injection of extracts of the diarrheal stools of rachitic children. Bland Sutton found that young monkeys deprived of mother's milk and fed on vegetables and fruit became rickety. Two young bears fed on rice biscuits and raw meat had extreme rickets. A lion whelp weaned early and fed on milk developed rickets. The lion cubs already mentioned, fed on raw meat, developed rickets

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on meat and were cured on milk, ground bones and cod-liver oil. It would seem from a consideration of these positive results that rickets may be produced by an improper food which may be too difficult of digestion or contain too little sugar and fat or too much starch. Also we have evidence that certain bacteria, perhaps intestinal, or intestinal toxins may produce rickets.

Comby, of Paris, considers rickets infrequent in infants nursed at the breast and in support of this statement notes, that in China, Japan, India, etc., rachitis is unknown. These countries to a great extent have in common another factor, a warm climate. Rickets does occur in breast-fed infants and efforts have been made to determine the defects in the mother's milk which produced the rickets. These analyses, however, have been made naturally after the child had well-developed rickets and were thus not analyses of the milk on which the child first developed the disease. Pfeiffer made seventeen analyses of mother's milk supplying 8 cases of rickets. He found no variation in this milk, in fat, proteid or sugar, from that supplied to other infants who did not develop rickets. He found, however, the phosphoric acid diminished one-half in some of the milk furnished the rachitic children but the change was not constant. These observations of Pfeiffer are so far as I know unconfirmed.

The following clinical observations have been made in New York:—

Dr. Eli Long examined in December, January and February, forty-six babies in the tenements of New York, varying in age from one to nine months with an average age of six months. These babies were fed on all sorts of food, breast milk, modified milk, Straus' modified, pasteurized milk, condensed milk and some on proprietary foods. Among these forty-six babies, many of whom had dirty and unhygienic surroundings, he found only 7 or 15 per cent. with marked rickets. Three of these were colored (there were only five colored children observed), and one an Italian (the only Italian child observed); of these 7 cases 3 were fed on condensed milk, one on condensed milk and breast milk, 2 on modified milk and 1 on modified milk and breast-milk. The small proportion of cases of rickets observed by Dr. Long may be accounted for by the early age of some of the children and by the fact that only marked rachitis was noted.

Of twenty-one infants varying in age from two to sixteen months and averaging eight months, carefully examined by Drs.

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Long and Steele, during April at the Pediatric Clinic of the University and Bellevue Hospital Medical School, they found that six showed evidence of rickets, mostly slight, or about 28 per cent. These children were from the tenements and represented the same class as those in whom in Boston, Dr. Morse found that 80 per cent. had rickets.

INDICATIONS OF RACHITIS IN THREE GROUPS OF BABIES.

	No.	Average Age.	Sweating.	Bald Spot on Head.	Rosary.	Pot Belly.	Small Thorax, Large Abdomen.	Delayed Dentition.	Craniotabes.
TENEMENT HOUSE.									
Bottle-Fed	13	4½ mos.	0%	77%	70%	46%	23%	....	8%
HOSPITAL.									
Breast and Condensed Milk	66	8 mos.	35%	76%	91%	57%	42%	16%	45%
Modified Milk	25	10 "	20%	56%	88%	44%	32%	50%	12%

I have carefully examined a number of children in two institutions for the care of children in New York. In one of these institutions I examined sixty-six babies varying in age from one to twenty-four months with an average age of eight months. The babies are in large nurseries with ample light and well ventilated during the day. These children are nursed, one woman supplying the milk for two babies. In addition, twice a day, the babies under four months of age receive a condensed milk mixture consisting of a one-eighth dilution of condensed milk with 3 per cent. of cane sugar added, so that the food apparently represents about, fat .9 per cent., cane sugar 9 per cent, proteid 1 per cent. Between three and six months they receive in addition to breast milk a condensed milk mixture with bread crumbs soaked in it which I have figured out to represent about fat .8 per cent, cane sugar 10½ per cent., proteid .9 + starch. Of these sixty-six babies, 23 or about 35 per cent. were noticed to sweat about the forehead; 50 or about 76 per cent. had a bald spot on the back of the head; 40 or 62 per cent. had coated tongues, and 1 had a geographical tongue. Of eighteen children of eight months or more of age, only 3 or 16 per cent. showed marked delay in dentition.

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Of the sixty-six babies, 60 or about 91 per cent. had an evident rosary; in 20 it was small; in 33 it was of fair size; in 7 it was large; in 7 the epiphyses were large; in 38 or 57½ per cent. there was the distended abdomen known as pot belly; in 28 or 42 per cent. this was associated with a small flaring thorax, giving the familiar appearance in rickets of a small thorax and large abdomen; 30 or about 45 per cent. showed craniotabes; 22 or 33 per cent. showed an anterior fontanel that was larger than normal.

It is interesting to note that the nineteen children in one of these nurseries that had been having whooping-cough showed no more evidence of rickets than those that had been free from this disease. In this institution with good general hygiene and immaculately clean, of babies fed for the most part on breast milk but supplemented twice a day by a condensed milk mixture representing the formula—fat .9, cane sugar 9 per cent., proteid 1 per cent., 91 per cent. showed evidence of rachitis.

On undertaking to examine children in another institution, where modified milk feeding is used, I noted first the condition of thirteen infants which had been received from the tenements, within a few days. They were rather young, varying in age from three months to nine months and averaging four and one-half months of age.

Of these thirteen children:—none showed sweating; 10 or 77 per cent. bald spots; 7 coated tongue; 9 or about 70 per cent, rosary, 6 small, 2 moderate in size; 6 or 46 per cent. pot belly; 3 or 23 per cent. small thorax and large abdomen; 2 enlarged bosses; 1 craniotabes. Of five of these babies fed on condensed milk all had rosaries; of 3 fed on modified milk one had rickets; of 2 fed on modified milk with gruels both had rickets.

Twenty-five children in this institution who had not been recently admitted, varying in age from three months to nineteen months, and showing an average age of ten months, had all been fed on modified milk.

Of these twenty-five children, 5 or 20 per cent. showed sweating about the forehead; 14 or 56 per cent. bald spot on back of head; 13 or 50 per cent. coated tongues. Of 15, 8 months and over in age, 7 or nearly 50 per cent. showed marked delay in dentition.

Of the twenty-five children, 22 or 88 per cent. showed a rosary; 3 showed enlarged epiphyses; 11 pot belly; 8 small thorax and large abdomen; 3 craniotabes.

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On comparing these statistics, we find that the tenement house children make a much better showing than those in hospitals, even when the hospital babies are for the most part breast-fed, and here we have data apparently at variance with the accepted ideas, for in one group of hospital babies we have ample light, good air, cleanly surroundings and mostly breast milk, with 91 per cent. rosaries; while the tenement house babies with bottle-feeding show only 70 per cent. rosaries. We cannot then agree with Morse, who examined for rickets 318 dispensary babies in Boston, and concluded, after a careful study of the nationality and food, that they had only one influence in common, and that was unhygienic surroundings.

CONCLUSIONS.

(1) Experiments on animals would seem to indicate that unsuitable food is capable of producing rachitis and that possibly certain bacteria or intestinal toxins may produce a similar result.

(2) The clinical observations are too limited for any positive deductions, but are interesting, inasmuch as they indicate less rachitis in the tenement than in the institution children fed on modified milk, and the greatest amount of rachitis in institution babies fed on breast milk with auxiliary feedings of condensed milk.

DISCUSSION.

DR. ADAMS.—I believe that rachitis is becoming more and more infrequent in proportion to the education of physicians in the proper modification of milk for infant feeding. The result of my observations in institutions is that we have very many less cases of marked rachitis, nowadays, than we had ten or fifteen years ago, and I have attributed it to this fact. The physicians are becoming more intelligent as to the methods of feeding infants; discarding the patent foods which almost invariably conduce to the production of rachitis, as well as other diseases.

DR. MILLER.—It seems to me that almost as important as improper feeding in cases of rachitis, is one of the factors that Dr. Freeman mentioned, namely, the want of sunlight and fresh air. In Philadelphia we have a great many colored people, almost as many as you have in Washington, and rachitis is very common among them. It is also frequent among Italians in our cities, who live in tenements and dark cellars, though they are practically getting the same food that they did at home, where they do not have rickets. I believe in the South rachitis is not very common among

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the negroes, because they get plenty of air and sunlight; while in Maine coast towns, where the children usually get plenty of fresh air and sunlight, though they are sometimes very improperly fed, either artificially or by mothers who are not able to properly nurse their infants, rachitis is very uncommon. It would seem, therefore, that this is a very important factor in the causation of rachitis.

DR. COTTON.—There is no doubt that a rachitic infant may be found at the breast, but I believe that all agree that it is extremely rare to find a rachitic infant at the breast where there is not a self-evident cause of its development. Examination of the hygiene in almost every instance reveals gross errors. Examination of the diet of the mother usually shows some marked deviation from normal dietetic hygiene. Examination of the mother's milk almost invariably reveals an unbalanced constituency. Infants at the breast of healthy mothers, giving normal milk, do not have rachitis. Why in this disorder we should constantly and positively close our eyes to the fact that the most relief is found in breast-feeding is a matter that I can only explain by stating that we have gotten into the habit of running away from this question of breast-feeding. If one tithe of the time and labor now spent on these other subjects of feeding and rachitis were devoted to the subject of lactation, its disturbances, the physiology of lactation and the hygiene of the mother, I believe relief would be found.

We know where to look for our rachitic cases. We go among the Italians; among the negroes; and occasionally you can find a case of rachitis at the breast; but if you will examine the hygiene and the diet of the mother, and examine the mother's milk, the cause will be very plainly seen. Take the mother, surround her with proper hygienic conditions, make her give good milk and I believe, even though she be changed from the warm to the cold climate, a predisposing factor, the frequency of rachitis will diminish.

I must say that by artificial-feeding there has been a great deal done. At times an infant has been taken from the mother's breast and fed on the best laboratory milk and it did well; but how much better to take the mother and see if we cannot make her give good milk before her next gestation. I believe that this would be an important factor in preventing, not only rachitis, but many acute intestinal disorders.

DR. SHAW.—Four years ago I was in Vienna when Prof. Monti was working on the etiology of rachitis. He found that in nearly every case there was a previous history of some stomach disorder. Examination of the stomach contents showed an increased amount of lactic acid and a diminished amount of hydrochloric acid. His theory was not, that the lactic acid produced the rachitic changes, but that they were influenced by the diminution of the hydrochloric acid. I think most of you have read

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Zweifel's exhaustive work on the etiology of rachitis in which he laid much stress on the diminished amount of hydrochloric acid in the stomachs of these cases. He showed the hydrochloric acid was dependent on the amount of sodium chlorid ingested, and that the nursing mothers in Saxony received very little salt in their food; hence, their nursing babies frequently showed signs of rickets. He came to practically the same conclusion as Prof. Monti through a different course of reasoning. For the past two years at St. Margaret's I have added salt to the food of every infant and it seems to me that we have had very much less rachitis than formerly.

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CONCERNING THE NATURE OF STILL'S TYPE OF CHRONIC POLYARTHRITIS IN CHILDREN.

BY DAVID L. EDSALL, M.D.,

Philadelphia.

A peculiar form of chronic joint disease in children was described in 1896, by Still, and is now widely known in connection with his name. The chief characteristics of this class of cases that Still believes distinguish it from arthritis deformans and from chronic rheumatism, as these latter conditions are ordinarily recognized, are marked enlargement of the lymph nodes, and usually of the spleen; a tendency to irregular, and at times rather marked, fever; the absence of noteworthy bone deposits; and the fact that this peculiar disease occurs almost exclusively in young children, usually appearing before the second dentition.

Still believes, and others agree with him, that the disease is probably an infection. The fever, the swelling of the lymph nodes and splenic enlargement, and the general course of the disease make it seem highly probable that this view is correct. The cause of the infection, however, has never been demonstrated.

The observations that I am about to present have led me to the belief that, in the individual case that I am recording, and perhaps in other cases, the condition is probably a peculiar form of chronic, widespread tuberculosis of the joints. Still's disease, of course, differs materially from joint-tuberculosis, as this condition is usually conceived of. There is no tendency to the production of white swelling, to abscess or sinus formation, or to the appearance of other destructive lesions of the joints; the post-mortem examinations that have been reported have not demonstrated any gross evidences of tuberculosis; and a very striking dissimilarity is found in the fact that Still's disease tends to involve most of the joints, while tuberculosis usually remains limited to a small number. The latter point, however, and the apparent absence of the lesions of tuberculosis are not extremely weighty arguments, when one considers that a form of lymphatic disease that shows no macroscopical or histological evidences of tuberculosis, and that is widely different from ordinary scrofulous lymph

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nodes, has been shown, chiefly by Sternberg, to be a not uncommon condition. Still's disease, with the joint-lesions left out, is, in its chief clinical characteristics, not at all unlike the Sternberg type of chronic lymphatic tuberculosis; and, with this fact in mind, the general clinical history of Still's disease is, in itself, sufficient to suggest the possibility that the disease is tuberculous.

The case upon which my observations have been made is that of a boy, now thirteen years old, an inmate of the Philadelphia Home for Incurables. He was admitted to that institution in March, 1901, and was first seen by me in July, 1902. Previous to entering the Home for Incurables, he had been in the Children's Hospital, under the care of Dr. Stengel and of Dr. F. A. Packard; and the records of that hospital have kindly been furnished to me. The child was under observation in the Children's Hospital from August, 1900, to March, 1901. The history given upon entering states that he had never been in very good health, and that he had been ill-treated and brought up in unhygienic surroundings. There was no tuberculous or rheumatic family history. He had measles three years previously; but had had no other illness until March, 1900, when the joint trouble began.

This had had a gradual onset, but had been associated from the beginning with failure in general health. It had first affected the right ankle, then the right foot, and next the left ankle and the left foot—this in the course of about three months. Subsequently, one knee after the other had become involved; and in July, the disease had involved both hands. There had been a progressive increase in the severity of the joint-condition with moderately acute exacerbations of the pain, swelling, and stiffness; but there was no history of severe acute illness at any time during the progress of the disease.

At the time of his admission to the Children's Hospital, it was noted that the patient's axillary lymph nodes were swollen and hard; the epitrochlear and the inguinal lymph nodes were palpable, and the splenic dullness was increased. At that time there was also swelling of the wrist joints and fluctuation over them. Most of the finger-joints were swollen, and a number of them were enlarged. The joints of both great toes were swollen and tender, as were the ankles and the region over the tarsus. The knees were enlarged, and movement at the knee-joints caused pain. The blood showed 14,000 leukocytes and 60 per cent. of hemoglobin; the red cells numbered 6,000,000. There was a systolic cardiac murmur

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in the pulmonary region, but no definite evidence of organic heart-disease.

While in the Children's Hospital, the boy had repeated temporary exacerbations of the objective and the subjective joint signs; and it was repeatedly noted that the lymph nodes in regions corresponding with the joints that had shown acute exacerbations of their disease had become larger. His temperature during this time was very irregular. He had a number of periods of persistent fever, ranging as high as 103°F., and lasting for several weeks. During the intervals the temperature was sometimes nearly normal, but sometimes showed irregular temporary elevations. The boy was sent to the Home for Incurables unimproved, with more extensive disease than when admitted into the Children's Hospital.

When I first saw him in July, 1902, his general condition had decidedly improved, as contrasted with the condition described in the notes of the Children's Hospital. The joints remained much the same as they had been upon admission into the Home for Incurables. For some months, the patient had had no exacerbation of the joint condition; indeed, he had had almost no symptoms referable to the joints, except the disability that the disease had produced.

His lymph nodes at that time showed striking enlargement. A large number of the anterior and the posterior cervical lymph nodes were palpable; they varied in size from small granules up to the dimensions of a pigeon's egg. There was marked enlargement of the axillary lymph nodes; those in the right axilla running up to a diameter of about an inch, while in the left axilla many nodes were enlarged, one being of the size of a small hen's egg, and several having a diameter of as much as an inch. The brachial and the epitrochlear lymph nodes were readily palpated, and a number of the inguinal lymph nodes were as large as hazelnuts. The splenic dullness was distinctly increased, but the spleen could not be felt; the boy's deformity, however, rendered it very difficult to palpate the splenic region.

There were no definite signs of pericarditis, a complication of this type of joint-disease that Still states is common. The patient likewise showed no signs of other disease excepting that of the joints, which was very extensive.

The main points about the joint condition were as follows:—The jaw was unaffected. There was marked involvement of

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the cervical spine; rotation of the head and anteroposterior motion being extremely limited. The left shoulder was uninvolved; motion in the right was decidedly limited, but there was no other palpable or visible change about the joint. Both elbows were nearly fixed at about a right angle. Movement in the elbow joint was very slight. The rotary movements of the radius were good. The right elbow was globose in appearance, apparently chiefly from thickening of the soft tissues of the joint. Both wrists were nearly rigid at about a right angle. There was swelling over the carpus and over the upper half of the metacarpus on both sides; and there was also some fluid over the right carpus. The carpal and metacarpocarpal joints were practically immobile. There was very little motion in the metacarpophalangeal joints on either side, all these joints being overextended. The first and the second of them on each side showed marked thickening, which seemed to be partly of the ends of the bones. The first interphalangeal joints of all the fingers were thickened, particularly on the left side; and this thickening seemed to be partly bony. There was but little motion in these joints, the position being one of flexion at about a right angle. The thumbs showed thickening of both joints, and subluxation at the metacarpophalangeal joint; and the joints were almost entirely fixed. There was also apparently slight bony enlargement of these joints. There was slight limitation of the motion of the left hip; the right hip was not involved. Both knees were globose; the ends of the bones were possibly slightly enlarged, and the soft parts were somewhat thickened, producing a partial obliteration of the normal prominences. There was some fluid under the left patellar tendon. The knees were held at a right angle, and the hips could be straightened but little. There was decided limitation of the movements of the ankle-joints; the joints looked somewhat globose; and there was swelling over the tarsus and the metatarsus, the enlargement being apparently of the soft tissues. Both the tarsus and the metatarsus seemed to be rigid. The toe-joints showed practically no involvement. None of the joints showed hyperostosis or bony grating.

The following is a skiagraphic report by Dr. H. K. Pancoast, who was kind enough to make a series of plates for me on May 4, 1903. Only the main points in connection with the joints that showed noteworthy lesions are mentioned.

Left Hand and Wrist.—Dense adhesions are present between all the carpal bones and in the radiocarpal articulation. The same

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is true along the line of the carpometacarpal articulations, especially between the first and the second metacarpals, and between the trapezium and the trapezoid. There is apparently a slight subluxation at the ulnar side of the first phalanx of the thumb. There are well-marked adhesions in the latter joint, as well as in the interphalangeal joint. Deposits are present in the other metacarpophalangeal joints, with a widening of the proximal epiphyses of the phalanges.

Right Hand and Wrist.—The radiocarpal, carpal, and carpometacarpal joints present a similar appearance to those of the right hand; but the adhesions are denser. The proximal ends of the first phalanges show a decided widening, corresponding with the epiphyses. There is a loss of bony substance in the distal end of the second metacarpal. The same end of the third metacarpal is apparently worn away on the radial side, where the proximal end of the phalanx is undeveloped.

Right Elbow Lateral View.—The olecranon epiphysis is possibly more advanced in ossification than is usual at this child's age. There are dense adhesions between the posterior portion of the olecranon and the region of the olecranon fossa. The elbow is flexed at about 120°F. There is no loss of bony substance at this articulation. There are a few adhesions between the coronoid and the anterior surface of the humerus on this side.

Left Knee, Lateral View.—There are well-developed adhesions between the patella and the lower femoral epiphysis, the patella being in relation with the latter only. The adhesions apparently involve both condyles. The remainder of the joint cavity is fairly clear. There seems to be a thickening of the articular surface of the upper tibial epiphysis. There is no apparent loss of bony substance.

Right Knee, Lateral View.—There are well-marked adhesions between the patella and the femur, and between the femur and the tibia; and the articular surface of the femur and of the tibia show very marked thickening, this being much more pronounced than in the other knee. There is a decided increase in calcification at the epiphyseal line in the upper end of the tibia as compared with the opposite side.

The Hips.—Both hips show marked adhesions between the femoral head and the acetabulum, and marked thickening of the articular surface of the femur. The angles of the necks seem to be more obtuse than is normal. The necks are greatly thickened,

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and on the left side the infiltration may readily be seen to involve the capsule.

Since the time of the general physical examination described above, the boy has improved slowly, but decidedly. He has gained flesh; he has a good color; and many of the joints have shown marked improvement in motion. In July, 1902, he was confined to a roller-chair; he is now able to run about on crutches and to walk a little without either crutches or cane. The chief improvement is in the knees and in the ankles, though the wrist-joints and some of the finger-joints show marked improvement. The boy has had practically no pain during the last year, except at the time of some tuberculin-injections, which will be mentioned.

The enlargement of the lymph nodes has decreased to a striking extent. Two or three nodes as large as peas may still be felt in the neck. All the other cervical lymph nodes have decreased so much as to be scarcely palpable. The lymph nodes in the right axilla have shrunk, the largest being now the size of a hazel-nut. Those in the left axilla decreased greatly in size, so that at the time of an operation that will be mentioned, the largest was about an inch and a quarter in diameter. The inguinal lymph nodes, also, seem to have become smaller.

During the whole of the year 1902, the boy had a tendency to occasional brief elevations of temperature, usually to about 100°F. This fever was not accompanied by other symptoms. During the last six months the temperature has been taken only irregularly; the patient has had but slight variations from the normal, so far as the records show.

With the suspicion already mentioned that the whole condition might be tuberculous, the child was subjected to tuberculin injections, with the following results:—

On October 29th, 1½ mg. was injected, without any definite result. On November 5th, at 3 P.M., he received 3 mg. On the 6th, at 3 A.M., the temperature was 101°F.; and at 6 A.M., 102°F. It remained at this point for six hours and then declined, reaching normal at midnight. The boy felt generally miserable; a number of the joints became very painful (while, as already stated, he had been free from pain for some months previously); and he begged to have his daily massage omitted, as the tenderness of his joints was so great that he could not stand having them manipulated.

Because the boy had, in ordinary circumstances, a tendency to

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fever, another injection was given on November 12th at 9 A.M., his temperature meanwhile having been regularly below 100°F. Six hours later, at 3 P.M., the temperature was 100⅝°F.; at 6 P.M., 103°F. It remained near 102°F., for twenty-four hours, and then rapidly declined to normal, remaining normal for four days, after which the regular temperature-records ceased.

During this reaction the boy experienced marked nausea, felt generally ill, and complained of intense pain and tenderness in many of his joints. The wrists and the ankles were swollen; there was some redness around them; and they were very tender. Movement of the joints caused the patient to cry out with pain. During the reactions the enlarged lymph nodes became slightly tender; but no other change in them could be noted. The boy showed no signs of pulmonary disease, either during the reactions or at any other time.

On March 28, 1903, Dr. Jopson, at my request, removed the lymph nodes from the left axilla. These were of different sizes, the largest being somewhat over an inch in diameter. Two were about three-quarters of an inch, and the fifteen others secured were smaller. Their macroscopic appearance was in no way striking. There was no suggestion of tuberculosis in their appearance, and their color and consistency were about normal.

Dr. Ravenel kindly made a microscopic examination of these lymph nodes and inoculated guinea-pigs with an emulsion made from them. There were no histological evidences of tuberculosis, and an examination of several sections showed no tubercle bacilli. The only histological changes noted were slight enlargement of the Malpighian bodies and poor staining of their cell-nuclei. An examination of the emulsion from several of the lymph nodes, however, showed a large number of acid-fast bacilli, having the typical appearances of tubercle bacilli, some of them lying singly or in pairs, while a number of fairly large clumps could be readily discovered. Five cc. of an emulsion, of a consistency just sufficient to pass through a hypodermic needle, was injected into each of two guinea-pigs. To our surprise, the result of these inoculations was entirely negative. The animals were still well five weeks after the inoculation, and they were then killed. In the absence of Dr. Ravenel, Dr. Gilliland kindly examined them; the most careful search failed to disclose any evidence of tuberculosis or of any other disease. Dr. Ravenel has no doubt that the bacilli seen were tubercle bacilli; and this cannot well be questioned, par-

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ticularly in view of the violent tuberculin reactions. Pseudo-tubercle bacilli so far as known do not invade the tissues generally; and this point, together with the tuberculin reactions, makes it seem practically indubitable that the bacilli were tubercle bacilli. The result of the inoculations indicates that the bacilli in the lymph nodes must have been either dead or of extremely low virulence. Dr. Ravenel thinks that they were dead, because they were present in such large numbers as to make it certain that so many must have been injected into each guinea-pig that it would scarcely have been possible for the animal to have resisted infection. It is quite possible that some of these bacilli were still alive but of low virulence. This point cannot, of course, be definitely settled.

These observations indicate that this boy has widespread tuberculosis, which is of such a low degree of virulence that he has been able to kill off large numbers of tubercle bacilli and, finally, to control the progress of the disease. I believe that the results also furnish strong indication that the disease of the joints is tuberculosis. The intense local symptoms in the joints at the time of the tuberculin injections; the fact that the enlargement of the lymph nodes was so distributed as to be in apparent relation with the joint disease, and that much at least, of the enlargement of the lymph nodes is known to have followed the joint disease; also, the fact that the nodes have several times been noted to be increased or decreased in size, in accordance with the progress of the joint disease—all these indicate that both the joint disease and the enlargement of the lymph nodes are probably due to the same cause, and that the disease of the joints has produced the node enlargement. Therefore, since the nodes are tuberculous, it seems highly probable that the disease of the joints is also tuberculous.

It may very properly be objected that the tuberculin reactions described were due to the tuberculosis of the lymph nodes, and that they indicate nothing concerning the nature of the joint disease. I, of course, admit the possibility of this, but I think it improbable, because of the relations already mentioned between the course of the lymph node enlargement and the progress of the disease of the joints; and also because of the violent symptoms and the objective signs in the joints at the time of the reactions. I am aware that joint pains not infrequently occur at the time of tuberculin injections; and that they are, naturally, most likely to occur when the joints are

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the seat of disease. I think, however, that the facts that I have stated make it highly probable that the joint manifestations were due to local reaction. The symptoms referable to the joints were far more violent than were those observable in the lymph nodes, the latter showing only a little tenderness and slight swelling. This makes it probable that the joints actually reacted, and were the seat of a much more active process than were the lymph nodes. The symptoms at the time of the reactions, therefore, as well as the clinical course of the disease in the joints and in the lymph nodes, favor the view that the disease of the joints was the essential condition, the disease of the lymph nodes being largely or entirely secondary. Absolutely definite proof of the nature of the joint disease can, of course, be given only through histological and bacteriological study of the joints themselves, in cases of this kind that come to necropsy, or in cases in which the excision of one or more of the joints might be a proper method of treatment. The facts that I have presented, however, justify, I think, a serious belief in the probability that this patient has a form of tubercular arthritis that is peculiar in its relatively benign local lesions, and especially in the involvement of many joints; and this indicates the strong possibility that Still's disease may, in a considerable proportion of instances, be of this nature; a possibility that, as stated, seems to be suggested by the chief clinical characteristics of this disease.

Tuberculosis is believed by Poncet and some of his pupils to be responsible for a large number of cases of joint disease that are not usually considered to be of this nature. I have discussed this larger question elsewhere.* The case of Still's disease referred to in that communication is the same as that reported in this paper.

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* Edsall and Lavenson, American Journal of the Medical Sciences, December, 1903.

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PROPHYLACTIC MEASURES AGAINST THE SPREAD OF VULVOVAGINITIS IN HOSPITAL SERVICES.

BY HENRY KOPLIK, M.D.,
New York.

One of the most annoying scourges of a children's hospital service is the vulvovaginitis of young girls, anything, therefore, that can be done to prevent an outbreak or the spread of this affection in hospitals, is of the greatest value and importance. Not only has this disease certain repugnant features but, with the vulvovaginal area once affected, the little sufferer becomes exposed to the dangerous consequences of the disease and a menace to others. There is no affection of infancy and childhood which resembles so closely the exanthemata in its manner of spreading from child to child, without leaving any clue as to how the spread of the disease is initiated, or accomplished. I think we may dismiss, without further cavil, the contention that the affection may spring up spontaneously in a children's service, although this is occasionally hinted at. There must be an acute or chronic case preceding an outbreak of the disease; in other words, there must be a source of infection. The exact method by which a healthy child is infected by another child suffering from vulvovaginitis, is still unknown. I might state that nothing in all the volumes that have been written on the subject gives us any positive data. One thing stands out prominently, that the mucous membrane of the parts affected in children of both the male and the female sex, is strongly predisposed to this infection, that is, the gonorrheal form of which I am speaking.

It has been supposed that the infectious material becomes implanted on the parts by the fingers of the nurse, or conveyed by the napkins, by the thermometers, the catheter, or, in fact, by any infected article. It is, however, certain, though strange, that a pure culture of the gonococcus rubbed on the parts will not always cause a vulvovaginitis. Epstein made these very experiments and failed to reproduce the disease. We must, therefore, conclude that the secretions themselves are in some way carried from patient to patient.

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In a children's service there are two forms of gonorrheal discharge, which are exceedingly prone to give rise to vulvovaginitis, urethritis, or proctitis. One is the vaginal discharge, the other is the rectal.

Of the vaginal discharge, which is more apt to be the starting point of an epidemic, the chronic scanty discharge is much more to be guarded against, because less evident, than the acute, profuse one, which is easily seen by a nurse. As an illustration, let me cite a case in point.

A child in my hospital ward, suffering from chronic cardiac disease, was found to have an acute vulvovaginitis. On her discharge from the hospital her vulvovaginitis was practically in the condition of the gleety stage of a gonorrhea. Some days there would be a very slight, thin, mucous discharge; at other times there was no discharge. This patient was readmitted three months later for the treatment of her cardiac affection. A careful examination of the mucus of the vagina failed to reveal any gonococci, but I directed that the child be carefully isolated. I have seen such cases show a profuse discharge, with the return of the gonococci, without, apparently, any provocation. We see the same manifestations in the adult subject suffering from apparently cured posterior urethritis.

Another case which illustrates how the utmost watchfulness must be exercised over these patients is that of a child of eight years recently admitted to my ward without a sign, on repeated and careful examination, of a vaginal discharge. After having been in the hospital two weeks a scanty mucous discharge appeared. Examination showed this discharge to be gonorrheal. Inquiry revealed the fact that this child had suffered from a vaginal discharge some time before her admission, which had disappeared to reappear weeks later. Had it not been for the system, by which each child in my service is practically isolated from its neighbor, these 2 cases would have been the starting points of two epidemics of the disease. Before the present system of isolating one child as much as possible from the others was instituted in my ward, a case similar to either one of the above was frequently the cause of infection in quite a number of other children.

The rectal discharges are none the less dangerous and any purulent discharge from the rectum of a boy or girl should be im-

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mediately examined for gonococci. This may seem extreme, but the following case, which is only one of many, will illustrate my point.

A boy, eight years of age, was admitted to my ward suffering from a lobar pneumonia with a slight amount of pleuritic effusion. A few days after admission it was noticed that the boy had a discharge of blood and mucus, at times mingled with pus, from the rectum. There was no pain but a slight rise of temperature, due to the pleurisy or pneumonia, caused the staff to think of a possible dysentery. I directed, however, that the rectal discharge be examined for gonococci and the boy isolated. The mischief, however, had been done and in a short time three children developed vulvovaginitis. The boy's rectal discharge was gonorrheal and had existed before admission to my service.

Another case is illustrative. A boy, four years of age, was discharged from my service convalescent from pneumonia. In four days he returned with a history of periproctitic abscess. He was admitted at night and in the morning when I was told that the patient had a rectal discharge I directed that he be isolated and the discharge be examined for gonococci. This was done, but in this case, also, the boy was the starting point of an epidemic in which seven girls in my service were infected with vulvovaginitis. This patient had left my ward well and without any rectal discharge. There was no case of vulvovaginitis in the ward during his stay. He returned to the hospital with his infection. In the uncertainty of the night nursing the infection of the other children occurred.

How is vulvovaginitis, proctitis, or urethritis conveyed from patient to patient? I have made this question one of close study and find that in a hospital service there are many avenues by which this affection may be communicated to the patients. All utensils, such as bed pans, catheters, douche nozzles and thermometers, are the first source of infection. A second prolific source is the linen and rubber sheets, the diapers, the underclothing, the wash rags and basins and, finally, though not the least, the bath tubs. During the past five months no case of vulvovaginitis or urethritis has arisen in my service, and I attribute this fact to the system of complete isolation of one child from the others carried out in my ward. Every child admitted to my service is now examined as to the presence of a vaginal, urethral, or rectal discharge. Not only are the girls examined externally but a spread is made of the vaginal

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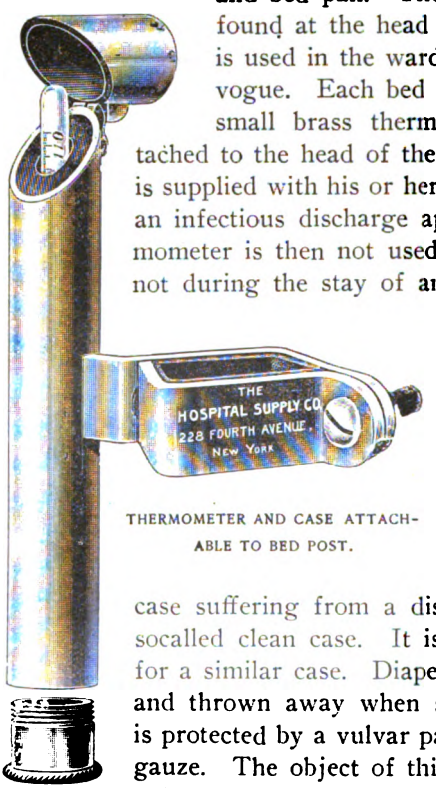
secretions. No child is bathed in the common bath tub until pronounced free from vaginal, rectal or urethral discharge by the house physician. If there is any suspicious discharge from the vagina, urethra or rectum the patient is isolated and put under certain restrictions to be mentioned. Every female child on admission to the regular ward is given an individual basin, comb and bed pan. The basin and comb are to be found at the head of each bed. No bar soap is used in the ward, liquid soap alone being in vogue. Each bed is fitted permanently with a small brass thermometer case (see cut) attached to the head of the bed and thus every patient is supplied with his or her own thermometer. Should an infectious discharge appear at any time the thermometer is then not used on other children, at least not during the stay of any little patient in the hospital. After discharging a patient the thermometer is carefully cleansed and placed for a time in 2 per cent. formalin solution. The thermometer case itself is boiled in soda solution for one hour. A bed pan once having been used on a case suffering from a discharge is never used on a socalled clean case. It is disinfected and used only for a similar case. Diapers are made of cheese cloth and thrown away when soiled. Every female child is protected by a vulvar pad made of several layers of gauze. The object of this is to avoid contact of the vulva with hospital linen. When hospital diapers were in vogue in my service vulvovaginitis was quite prevalent, when they were discarded vulvovaginitis became at once infrequent. I therefore believe that the ordinary laundry washing does not sterilize the linen, it only cleans on the surface and the steam does not reach all the interstices of the fabric. Every child in my service is supplied with its own utensils, and no utensils, such as thermometers, catheters, combs and basins, are kept in a common room where they are likely to become mixed, but they are placed at the bedside ready for use. The thermometer is,



THERMOMETER AND CASE ATTACHABLE TO BED POST.

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after use, cleansed carefully and replaced in its own case at the bottom of which is a formalin tablet, the fumes of which constantly bathe the instrument.

If a patient is found affected with a vaginal discharge the bed is surrounded with a red bandage to warn the nurses of the presence of an infectious discharge. The bedding, rubber sheets, draw sheets and all the linen of the patient are marked V. D. (vaginal discharge). This linen is never used except in a similar case. Soiled linen is first sterilized in the hospital sterilizer before going to the general laundry. Bed pans, catheters, nozzles and douche apparatus are all marked with the name of the patient and kept apart from similar utensils in the ward. The nurse who attends the patient is not allowed to take the temperature or touch the genitals of any other patient in the ward. It will thus be seen that I have instituted a most complete system of isolation by which each little patient in my service is protected from fellow patients. Since this system has gone into use, should a vulvovaginitis or rectal discharge appear it cannot spread easily, if all the various safeguards that I have marked out are maintained. It may seem to some that I have been at too much pains concerning a very simple matter. Since, however, I have seen several deaths result from an infection of the peritoneum resulting in this disease, I have been satisfied in my own mind that my studies were not in vain. Moreover, the very evident nonchalance with which some physicians look upon vulvovaginitis is simply a reflection of their lack of knowledge of the serious complications and sequelæ of this affection. Years ago Sanger pointed out that many cases of salpingitis in young girls and sterility in later life could be traced to this infection in early childhood. Vulvovaginitis, therefore, if it arises in a hospital service and spreads in epidemic form only proves to my mind a laxity in precautions necessary in every service devoted to the treatment and management of diseases of infancy and childhood.

692 MADISON AVENUE.

DISCUSSION.

DR. CHAPIN.—We take a culture from every female child before entrance to the Babies Wards of the Post-Graduate Hospital, and we have found that in many cases in which we had not suspected specific vulvovaginitis, it does exist. So, I think the

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precautions suggested by Dr. Koplik are very valuable. It is the bane of hospitals for little children that such things exist, and they happen much oftener than we think, for some clinical manifestations are very difficult to make out. We have sometimes found, on close examination, that the parts which showed very little evidence of vulvovaginitis, had the gonococcus present.

DR. COTTON.—I am very much interested in Dr. Koplik's paper because recently I had a very trying experience in the Children's Ward of the Presbyterian Hospital. Our service is divided into quarters, my quarter beginning in July and extending through August and September. During that time the prevalence of typhoid fever kept the ward, which would accommodate about twenty-five patients, pretty full. There were 16 or 17 cases of typhoid at one time. No case having a discharge from the vagina or urethra occurred during my service of the three summer months, with a single exception of a surgical case reported to be a case of rectal ulcer. When I returned to the service three months later the ward was quite full of vulvovaginitis cases. In all we had 14 cases; but we have gotten rid of it.

The remarks of Dr. Koplik have proved particularly interesting to me. My colleague, Dr. Dodson, who had charge of that ward during most of the time that this infection developed, is present and I should like to ask permission for his experience at the beginning of that epidemic.

DR. DODSON.—I assure you I appreciate the courtesy of the invitation, and I have been especially profited by Dr. Koplik's paper. It is of great importance that attention should be called to this infection of vulvovaginitis in children and to the necessity of taking extreme precautions, for I judge from other physicians in charge of wards and children generally, that the importance is not fully appreciated and it is not at all uncommon to look upon the infection as a trivial affair.

One who has seen a few cases of gonorrheal peritonitis develop from vulvovaginitis and appreciates the disastrous consequences that are very apt indeed to follow from such an infection, can certainly not fail to realize the importance.

Two or three things occur to me from our experience in the Presbyterian Hospital, and one is that in this matter we are absolutely at the mercy of the nurse. It must be in the vast majority of the cases through the carelessness or lack of knowledge of the nurse that this infection is spread, and that calls attention to the importance, as it seems to me, of a somewhat better plan of training nurses than that now in vogue. Great as is the improvement in the character of the nurse now from that of fifteen or twenty years ago, there is still much room for improvement, and I think one of the principal defects is to fail to train the nurse in the fundamental principles on which her art is based, especially that of the bacteriology. It seems absurd that we should expect the nurses to learn bacteriology from a few lectures without any prac-

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tical experience, and I believe that we may never have this corrected until the nurse is given from six months' to a year's training in a laboratory before beginning her work. After she attempts a few cultures she begins to realize the importance of care.

Then the disinfection of the ward is frequently left to incompetent persons. It seems to me that there ought also to be in connection with every hospital some person who does all of that sort of work in a thoroughly scientific way and is responsible for it; who knows the exact amount of formalin gas per cubic foot of space and is perfectly certain that the windows and doors are properly sealed. The subject is one of very great interest.

We found that the usual injections are not sufficient. The remedies for the cure of such a discharge must be kept in contact with the parts almost constantly.

DR. DORNING.—In searching for the cause of a vaginal discharge, in some cases, in addition to taking a specimen for a culture, it is well to examine further for a foreign body in the vagina. I have seen a number of cases in which that accounted for the discharge. One case in particular, which had gone through the hands of four very excellent practitioners, showed the presence of a corroded bone button which had been removed from the child's under garments.

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THE PROPHYLACTIC USE OF DIPHTHERIA ANTITOXIN IN SCHOOL CHILDREN.

A PROPOSITION TO IMMUNIZE YOUNG SCHOOL CHILDREN ONCE OR TWICE DURING THE SCHOOL YEAR WITH DIPHTHERIA ANTITOXIN AND THEREBY LESSEN THE MORTALITY FROM PRIMARY DIPHTHERIA, SCARLATINA AND MEASLES COMPLICATED WITH DIPHTHERIA.

BY AUGUSTUS CAILLÉ, M.D.,
New York.

The curative and immunizing properties of diphtheria antitoxin are established facts. According to recent careful and unbiased investigations, the mortality in primary diphtheria has been reduced two-thirds and the protective power of diphtheria antitoxin extends over a period of from three to six weeks or more. With an agent so powerful for good at our command, the question naturally arises: Do we or do we not make the best use of our new therapeutic acquisition?

It is not the intention of the writer to discuss the indications for the use of diphtheria antitoxin, but to suggest a new use for this agent, by advocating an immunizing injection for young school children once or twice during the school year, for instance, in November and February, with the hope of preventing infection from primary diphtheria or croup; and, furthermore, with the hope of lessening the mortality of the severe forms of scarlatina and measles, a large percentage of such cases being complicated by diphtheria from the beginning or in the course of the disease.

It is well known to the experienced medical practitioner that cases of scarlatina which show a complicating diphtheria from the onset are of a very grave type. In such cases we often observe an overwhelming sepsis with delirium and circulatory failure. In scarlatina with complicating diphtheria setting in after the first week, the septic symptoms are never so acute and urgent.

In measles we have diphtheria as an early or late complication but the most important diphtheria complication of measles

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is diphtheritic croup. The mortality from scarlet fever plus diphtheria and measles plus diphtheria is quite high, and the writer is of the opinion, based on clinical experience, that this mortality can be markedly reduced by means of protective inoculations of diphtheria antitoxin. Such prophylactic management will have no effect upon pure and simple scarlatina or measles, but will certainly create more or less immunity as regards grave diphtheritic complications; or, in other words, turn a grave disease into a milder disease.

During the past two years the writer has practically followed out this line of thought and has immunized a number of school children twice during the school year. No figures are given for no one can tell what might or might not have happened. It may be stated, however, that not one of the children (altogether about twenty) contracted primary or secondary diphtheria and in no case was there the least unpleasant or unfavorable reaction after the protecting injection. The parents readily accepted the "protecting vaccination for diphtheria," and the writer has every confidence in the feasibility of this plan for communities in which diphtheria is endemic or epidemic.

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THE VALUE OF THE INCUBATION PERIOD IN THE DIAGNOSIS OF THE ACUTE EXANTHEMATA OF CHILDHOOD.

BY ALFRED HAND, JR., M.D.,

Philadelphia.

Strictly speaking, the incubation period of a disease is the time which elapses from the invasion of the body by the specific germs to the initial symptoms of an attack of the disease. It is necessary to qualify this by adding that that invasion only is to be considered which results in an attack of the disease, for it is likely that human beings are frequently subjected to repeated invasions by pathogenic germs, all but the last invasion of which are overcome, and it would be manifestly illogical to give as the period of incubation the time from the first invasion (if it could be determined) to the outbreak of the disease.

The duration of the incubation period probably depends on two factors, the virulence of the invading germs and the vital resistance of the individual. To certain diseases, however, the susceptibility of the human race is so great that when an individual unprotected by a previous attack is exposed to the contagion of one of these diseases, an attack of that disease is very likely to occur and the time which elapses from that exposure to the outbreak is, from the clinical standpoint, the period of incubation.

It is not necessary to refer to the reasons for the greater prevalence of some of these diseases in childhood. Three of them, measles, scarlet fever and rötheln, are the ones which I wish to discuss with reference to the value of their incubation periods in the diagnosis of these diseases. This value is largely a matter of personal opinion and the individual practitioner's estimate of it depends partly upon his early instruction, partly upon personal experience and partly upon the experience of others. In reporting an instance of unusual length for the incubation period of scarlet fever, I have added a review of the statements on the subject by various writers, hoping that something of value might appear.

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THE INCUBATION PERIOD OF MEASLES.

The opinions on this come nearer to harmony than for either of the other diseases. Comby, Henoch and Bendix give 10 days as the definite period with 13 or 14 to the eruption; Starr and Ashby and Wright give from 10 to 12 days; J. L. Smith, D'Espine and Picot say 12 days, the latter giving 14 days to the eruption as does Unger; Holt gives a table of cases, 66 per cent. of which were from 11 to 14 days; Rotch and Goodhart agree, giving 10 days as the usual period, with a possibility of 3 weeks; Dawson Williams says 14 days to the rash, but gives variations of 7 to 18 days; Baginsky gives nearly as short a lower limit, 8 days, with 10 as the usual and 14 as the maximum, while Carmichael gives the lower limit as 6 days. Thus, only three writers give a longer period than 14 days, and three a shorter one than 10 days; so, if 3 days are allowed for the preëruptive stage, then the agreement on 10 or 11 days is nearly unanimous.

THE INCUBATION PERIOD OF SCARLET FEVER.

Using the works of the above-mentioned authors, a much wider discrepancy is found for this disease than for measles. The opinions are as follows:—Hatfield (in Starr's Text-Book), under 5 days; Holt, a table, 87 per cent. of the cases being between 2 and 6 days; Rotch, "uncertain and irregular," usually less than 7 days, often 2 to 4 days; Williams, 2 or 3 days, rarely 7; Goodhart, under 7 days, may be less than 2; Ashby and Wright, 2 to 5 days; J. L. Smith, from less than 24 hours to 6 days usually, but he has seen 12 days; Carmichael, 24 hours to 7 days, rarely longer, but he has seen 2 cases 2 weeks after returning home; Unger, 2 to 7, rarely 11 to 14 days; Sevestre (D'Espine and Picot) 4 to 5 days, rarely over a week; Moizard, 7 hours to 17 days; Comby, 1 to 3 days, rarely 7 to 8; Henoch, 4 days or less; Bendix, 3 to 6, rarely 9 days; Baginsky, 7 hours, seen by Thomas, to 20 days, seen by Hagenbach, Loeb, Torday and others.

THE INCUBATION PERIOD OF RÖTHELN.

This is, if possible, even more uncertain than that for scarlet fever. Bendix, Baginsky, D'Espine and Picot, Ashby and Wright and Rotch give 14 to 21 days as the limits; Carmichael and Williams, 12 to 21 days; Comby, 15 days, hard to determine; Boulloche (in Comby's Traité) says under 10 days to be taken with reserve, 18 to 20 days rarely, while 12 and 15 days have been

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determined exactly in some cases; Unger, 4 to 25 days; J. L. Smith, from less than 7 to 21 days; Plant (in Starr's Text-Book), 7 to 21 days; Holt, 5 to 22 days.

From the above it seems that in measles the vital resistance of the individual plays but a minor part in warding off an attack; the high degree of its contagiousness being a further illustration of this, and it would also seem that the germ has a definite period of evolution analogous to that of the organism of malaria. It also seems that in scarlet fever and rötheln the virulence of the germ and the vital resistance of the patient influence, or rather actually determine, what the length of incubation shall be, and such variations are thereby produced as to render the incubation period of practically no value in the diagnosis of these diseases. More than this, if value is attached to it, we may be led to erroneous conclusions, for in given cases the periods may be identical for the different diseases.

The cases that I wish to report in illustration are the following:—In a family including three children, the youngest, Lillian, four and one-half years old, contracted diphtheria and was sent to the Municipal Hospital on November 22d, returning home on December 18th with a hemorrhagic nephritis which was months in subsiding; she looked to be in good health, the skin being exceptionally smooth and with no traces of desquamation, the nephritis being the only feature which aroused suspicion later that there might have been an overlooked scarlatinal infection either before or during her stay in the hospital. On my visit to her on December 31st I was asked to look at Theodore, aged six years, who was in the same room, his mother having just started to give him his morning wash and her attention being attracted by a rash on his neck and shoulders; an examination showed a diffuse erythema extending over the cheeks, neck, arms and trunk, the temperature being 98.5°F., the pulse 100 and no vomiting having occurred; the boy was isolated and the diagnosis reserved until the next day when, although the temperature was not elevated, the diagnosis was made of scarlet fever based on the general distribution of the rash, which was more intense and was clearly punctiform, the strawberry tongue, the rapid pulse 135, and the occurrence of vomiting, which took place after my visit on the preceding day. As we could not prove that Lillian had not had scarlet fever, and fearing the result of an attack with the existing nephritis, it was thought best to remove Theodore to the hospital.

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On January 16th I was called to see Harold, eight years old, who was in the grasp of a very severe attack of anginose scarlet fever, the rash extending from the scalp to the palms and soles. As before, we endeavored to protect Lillian by sending Harold also to the hospital, but he could not be admitted because Lillian had started in a week earlier with a classic attack of measles (coryza, dry cough, high fever, remission, Koplik's spots, and a typical eruption with high fever) and the accommodations of the hospital were insufficient to receive him without exposing a ward of scarlet fever patients to the possibility of an epidemic of measles. The brunt of the nursing fell upon the mother who was forced to go repeatedly from one child, still sharply ill with measles, to the other seriously sick with scarlet fever. The children were kept in separate rooms and disinfection practiced as carefully as possible; Lillian not contracting scarlet fever from this second exposure (a point in favor of the view that her nephritis was post-scarlatinal) and Harold not developing measles. The convalescence of each child was quite regular; Lillian having an almost imperceptible branny desquamation in a week, while Harold peeled in great flakes for about a month.

On the first of March Theodore returned home, having been detained in the hospital with an eruption of chickenpox, and the family moved to another house, hoping to have less sickness; but they were destined still to be interesting from a medical standpoint. On March 18th, Theodore was reported to have some rash and I expected to find measles, but, with a little hesitation, I diagnosed rötheln, and sixteen days later this was strengthened by both Harold and Lillian, who presented as clear signs of rötheln as I have ever seen. The diagnosis of rötheln in Theodore was further strengthened, practically, to complete confirmation one year later, when he had genuine measles, with coryza, Koplik's spots, somnolence, swollen face, fever and characteristic rash.

The strongest argument in favor of Lillian having had scarlet fever is the nephritis, and if we suppose that she did, then we must admit, as I believe everyone is willing nowadays to do, that the contagion can be spread by a patient by other means than the desquamation, probably by the secretions of the upper air passages or by the urine. The argument that she must have had it, or else she would not have contracted it from one of the two exposures is weakened, but not annihilated, by the low degree of contagion of the disease, and also by the clinical fact that members of the same

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family do not contract scarlet fever so easily from one another as from outsiders. This has been explained by the theory that the chemistry of the cells in the members of a family is somewhat similar, and when the germs of scarlet fever pass through one member of the family, they are so modified as to be incapable of setting up their pathogenic action in another member of that family. However, this does not always hold, so we may still reasonably return to the original contention that Lillian probably had had scarlet fever; that thirteen days after her return home Theodore developed distinct scarlet fever and that seventeen days later, Harold came down with unmistakable scarlet fever. To illustrate my point a little more clearly, if the diagnosis of Theodore's feverless attack had been rötheln, then at a period after that, corresponding to the usual incubation time for rötheln, Harold developed a rash; if Harold had only been mildly ill it would have been very easy to have called his sickness rötheln and much harm might have been done.

With regard to measles with its more distinct period, my own opinion is that this may often be of value in small family epidemics, helping to tell when the other members will develop the disease, but beyond this there is little value to be attached to it.

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PHARYNGEAL CROUP RELIEVED BY NASAL INTUBATION. (SOFT RUBBER TUBES.)

BY W. P. NORTHRUP, M.D.,
New York.

It happens to all to find infants breathing with great difficulty, because of acutely swollen tonsils and nasopharynx. Obstruction resulted in the case here related in dyspnea, which prevented the baby from nursing and from sleeping and had exhausted it to the point of threatened collapse. To quote the words of the hospital history: "Dyspnea urgent; child greatly exhausted; unable to sleep on account of dyspnea, although completely worn out."

The present case furnishes a familiar picture to the intubationist: Dyspnea urgent; recessions; a cyanotic, leaden color; stupor, restlessness, a sinking off to sleep, apnea, sudden starting, inspiring by voluntary effort, gasping, sinking again into unconsciousness.

It was evident that the child could not live long. There were the recessions of laryngeal obstruction and the familiar signs and symptoms calling usually for intubation. In this case the acute swellings of the tonsils and nasopharynx made it apparent that unless air could be conducted to the larynx nothing would avail from intubation. The main point in differentiating pharyngeal from laryngeal obstruction was the fact that the child when awake could open its throat by voluntary effort and relieve the obstruction. The nares were swollen quite shut, the pharyngeal tissues were so swollen that in sleep they fell together and left no room for entering air.

Nothing exhausts a child more rapidly than the combination of fever, lack of nourishment, loss of sleep and dyspnea. Unless its breathing could be made easy, this child had but a few hours of life in prospect. How was this to be accomplished? Answer: by tracheotomy or other means less heroic.

Case under consideration: A baby of five months, breast-fed, well nourished, well until present illness (December 16, 1902, Foundling Hospital); had been sick two days before entrance to hospital; had difficulty in nursing. On entrance: a profuse mucopurulent discharge from nose; grayish-white spots of exudate.

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probably diphtheritic, on both tonsils. Tonsils much congested. enlarged, meeting in median line; could breathe only with great effort. Besides the swollen tonsils, there were signs of bronchitis, coarse mucous râles and harsh breathing. After syringing the mouth, the baby nursed for the first time that day. During the following day it did not sleep and was nearly exhausted. Dyspnea was urgent; syringing of mouth and nose gave no relief. Nose was more obstructed than on previous evening; recessions very marked; steam inhalations gave temporary relief. At rounds on the following day, it was evident that dyspnea would soon completely exhaust the infant. There was obstruction, but it was not laryngeal. It was believed that intubation would not relieve the symptoms; tracheotomy was considered to be the only avenue of relief.

It occurred to me that air conducted past the swollen velum and tonsils would get to the larynx behind these tissues. As this was our first effort in nasal intubation, we were obliged to experiment. Two firm-walled bits of drainage tube, about the size of the small catheters used in nasal gavage, were selected. The tissues in the nose were so swollen and filled with mucopus that it seemed rather doubtful whether such small calibred tubes would resist the pressure and free themselves of mucopus, and would conduct air so far as to the larynx. At first they did not. After a short pause, bubbles of air came from the outer end of one tube, and soon the baby seemed a trifle relieved. By pushing the other tube in deeper, then slowly withdrawing it, we found the proper length of tube; two inches was about the length. To our satisfaction the baby soon sank into a quiet sleep of one hour and twenty minutes. It then nursed for twenty-five minutes, seemed quite relieved and doing well. That night it slept seven hours.

On the following day, the tubes were removed, and remained out from 10:15 A.M. till 5 P.M. At this time, dyspnea required their reinsertion. The baby slept two and one-half hours. When the ends of the tubes were pinched and occluded the child awoke at once, cyanosed. While the tubes were in place and pervious, the child slept or nursed. The baby subsequently developed pneumonia, from which it died. The slight exudate on the tonsils was found to be due to bacillus diphtheriæ.

RÉSUMÉ.—An infant five months old, after two days of illness developed such a severe condition of swollen membranes and enlarged tonsils, with nasopharyngeal obstruction, that it could not

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nurse, could not sleep. Exhaustion threatened; tracheotomy seemed the only resource. Nasal intubation with soft rubber tubes relieved the dyspnea, the child slept seven hours and nursed twenty-five minutes. The tubes were stiff-walled drainage tubes, two inches long. The infant later died of pneumonia, but intubation had fulfilled its function; it had tided the infant past the acute stage of pharyngeal swelling.

All intubationists have been called to intubate children who seemed to be developing croup, but who subsequently developed pneumonia. When I look back over my experience, I am sorry that I did not think to try, before, this simple method of relief of pharyngeal croup.

It is of passing interest to note that this nasal intubation was carried out at my suggestion by the present house physician of the Foundling Hospital, the son of my late colleague, who bears his father's name, Dr. Joseph O'Dwyer.

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O'DWYER INTUBATION INSTRUMENTS. ADDED SMALL TUBES FOR INFANTS UNDER ONE YEAR. EXHIBITION OF MODERN COMPLETE SET.

BY W. P. NORTHRUP, M.D.,
New York.

My purpose in presenting this subject and these instruments to the American Pediatric Society is to familiarize the members themselves with the appearance of the latest and best O'Dwyer tubes, so that when a spurious set comes before them, they may recognize it.

There are two kinds of intubation tubes offered for sale to the inexperienced. There are O'Dwyer's tubes and there are maker's tubes. Those I have to show you are O'Dwyer's tubes. The tubes themselves conform exactly to the model which O'Dwyer left us. These tubes are O'Dwyer's tubes with one or two added mechanical aids, worked out by his instrument maker and approved by the pupils of O'Dwyer.

They are, first, the fixed obturator. Instead of the obturator being screwed to the shaft of the handle just before it makes its right angle turn, it is continuous with the shaft. Each tube, as it lies in the box, has its obturator and shaft continuous. The obturator is short; the shaft bent at right angle is twice as long. In changing one tube for another the tube, its obturator and shaft go together. The shaft passes into a hollow handle and is caught and held by a thumbscrew. The object of all this will be quickly appreciated by an experienced intubationist. The tube will no longer (as has so often happened) turn at the screw and enter the larynx crosswise. This fixed (not unscrewing) obturator constitutes a real improvement, and no one would appreciate the suggestion more than Dr. O'Dwyer.

The second improvement.—Two new sizes of tubes are added for babies under one year. It occasionally happens that one wishes, in severe dyspnea, due to catarrhal swelling or in protracted, frequent and severe paroxysms of whooping-cough, to give temporary relief by intubation. Convulsions in whooping-

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cough may suggest temporary intubation. These two smallest sizes have been added to the sets at the Willard Parker Hospital, and at the Foundling Hospital. I have the small sizes and a set has been ordered by Prof. Massei, of Naples.

George Ermold, the instrument maker who made all of Dr. O'Dwyer's tubes, has made this set and another is ordered, at my suggestion. It contains all the improvements and is complete.

As I have stated, my object in presenting these instruments is that as many as possible may become familiar with the appearance of the O'Dwyer's tubes. In my opinion most of the tubes for sale in America, France and England are calculated to bring discredit upon the operation. I call upon the members of this Society, for the sake of protecting the repute of a most useful operation and one emanating from one who was an honored president of the Society, to do all in their power to encourage the use of O'Dwyer's intubation tubes. They are made by but one man.

The faults of maker's tubes are in the shape and finish of the tubes themselves. First, they do not fit the anatomy of the parts. Second, their lower ends injure the trachea. Third, their bulge is different and in this respect they differ from each other. Fourth, the collar and the top are worst of all. They do not fit the parts and cause pressure ulcers. All hooks and bales for facilitating removal, induce accumulations of thick mucopus and obstruction, beside causing ulcers.

It is enough to say of these tubes they are born of economy in making, of ignorance of anatomical relations, and of ignorance of pathological changes.

O'Dwyer's tubes were worked out through dead-house observations; maker's tubes were worked out in the shops by mechanics. O'Dwyer would not patent them and so protect them. Mechanics labor under no restraints. To manufacture cheap tubes to suit instrument sellers is an easy task. You will note that metal in the tubes is the maker's choice. Hard rubber is the choice of O'Dwyer. Hard rubber tubes involve a large initial outlay for steel moulds. A badly fitting tube of metal, incrusted after three days of wearing with lime salts, yielding to the motions of swallowing, embedding itself more and more in tissues swollen and diphtheritic, may cause much destruction of tissue.

Good results in intubation depend upon good tubes and good technique.

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DISCUSSION.

DR. MORSE.—Three years ago I saw in consultation with Dr. F. C. Cobb, of Boston, a baby two and a half months old with syphilitic rhinitis. The baby was in the condition described by Dr. Northrup and had a temperature of 104° F. It seemed very doubtful if the baby would live through the night. Dr. Cobb introduced soft rubber tubes into the nose in the way described by Dr. Northrup. They undoubtedly saved the baby's life. I have also found that in this nasal obstruction in babies an adrenalin solution usually relieves them temporarily, and sometimes tides them over a critical period.

It seems to me that there is room for an improvement even in this intubation set, and that is in the extractor. I have found in working upon infants that there is not room in the roof of a baby's mouth to get the extractor in properly. It is impossible to get the end of the extractor in line with the tube. It is always at an angle. I therefore had an extractor made with a shorter beak which does not take up quite so much room, and allows more space to work in. This has proved satisfactory.

DR. KOPLIK.—I think this is a very interesting communication of Dr. Northrup's. I would like to call attention to the fact that in young infants, if there is any nasal obstruction, the child has a tendency to swallow its tongue, even if no inflammatory process be present.

I have also tried the adrenalin solution Dr. Morse has referred to, and have obtained some relief. Some physicians have gone as far as to take out the tonsils in sick children suffering from pneumonia with a temperature of 104° F., an operation that one would hesitate to perform under ordinary circumstances, and for this reason I am very much pleased that Dr. Northrup has brought forth such a simple method of relieving the child.

As to the new form of intubation tubes, I have used the perfected instrument with the exception of the small tubes, ever since they have been out. They are certainly a great improvement over the old tube.

DR. CHAPIN.—I would like to call your attention to the fact that quite a number of years ago a specialist in New York, Dr. Goodwillie, devised a tube of flexible rubber which he made to fit in the inferior meatus. I have tried these tubes in a few cases, but never in the line that Dr. Northrup has suggested.

DR. DORNING.—I would like to ask if there is any objection to the little loop of wire on the top of some of the intubation tubes that are in the market? It seems to me that it renders extraction much more easy. I have used it in a number of cases and found that I could extract the tube more easily than with the extractor.

DR. NORTHRUP.—The objection to the wire loop is a very great one. It irritates the soft part of the epiglottis. There is also an

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objection to the foreign-made tubes which cause holes to be eaten into the esophagus and epiglottis. You need only look at these cheap tubes to see that what I say is true. The makers do not take the trouble to make the head of the tube correctly. They say it is all nonsense; that it is the fad of the doctors who only know intubation. They say that O'Dwyer was fussy. I beg to say that if this Society would hold to the original type it would do some good.

DR. SAUNDERS.—The same extractor will not do, in my hands at least, for children of all ages, and for several years past I have carried two extractors, one short and one long.

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A STUDY OF TWO CASES OF INTUSSUSCEPTION. (BOY SIX YEARS, GIRL NINE YEARS OF AGE.)

BY FRANCIS HUBER, M.D.,
New York.

In acute cases, with classical symptoms, a diagnosis of intussusception does not, as a rule, offer any great difficulty, provided, of course, the possibility of its occurrence be kept in mind. In discussing the subject it is well to recall two facts, first, the most frequent cause of acute intestinal obstruction in infants is the lesion now under consideration; second, the diagnostic symptoms are due to the existing obstruction or strangulation. A study of a series of cases shows that the younger the child the earlier symptoms develop.

In older children the picture is less pronounced. Invagination in them does not necessarily produce, or give rise to, early obstruction and strangulation, consequently the characteristic signs are more or less modified or even delayed. In chronic cases acute manifestations are absent. In fact the entire process is characterized at times by "extreme latency of symptoms." The presence of a tumor, possibly some slight hemorrhages at intervals, extending over long periods, may be the only evidences. Cases are reported in literature in which a tumor could be mapped out and in which pains recurred in paroxysms at varying intervals for months and months. Occasionally vomiting would occur. At no time, however, was there any interference with the action of the bowels, nor was any blood, mucus, or other abnormality, noticed in the movements.

The anatomical variety which favors a chronic course, is that in which the apex of the intussusceptum is formed by some part of the cecum, the cecocolic form, or when the colon itself is involved.

One form of the socalled prolapse of the rectum is practically a chronic intussusception of the upper into the lower part of the bowel.

Within a period of a few months, 2 rather unusual cases have been met with in practice. In the first, the diagnosis was not

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made until after the abdomen was opened. In the second a positive diagnosis was arrived at and confirmed at the operation.

For the following history I am indebted to the attending physician, Dr. Joseph Huber.

CASE I.—Nathan U. seen for the first time Sunday, Jan. 25, 1903. (The prior history having no bearing on the present illness, is therefore omitted.) The child looked well; temperature was 99° F.; he complained of pain in the abdomen; the pain was paroxysmal in character and not localized in any special area. The mother was positive in her statements that the bowels had moved twice daily up to the previous twenty-four hours. Codein. ⅛ grain hourly, high injections and cold or warm applications were advised.

January 26th; restless night, temperature 99° F., same paroxysmal pain, slight resistance of right rectus, bowels moved twice after injections, A.M. and P.M.

January 27th; temperature 99.6° F., condition the same; restless with pain in paroxysms all night.

At 11:30 A.M. I saw the boy in consultation for the first time. General condition excellent, pulse good and about 80 to the minute. Respiration natural and not accelerated. Tongue clean, no odor to breath. Heart and lungs negative. Abdomen: both recti tense, no pains in the right iliac fossa; palpation negative, as was also examination per rectum. No retention of urine or delay in the urination. No evidence of shock or collapse. The patient insisted upon getting out of bed to use the chamber. Between the paroxysmal pains he would play with his toys and took great pride in blowing a fish horn. No blood or mucus in the stools at any time.

Symptoms.—Pain referred to the umbilical region and tenderness in this area were constant. Pressure over McBurney's point negative. Colicky pains recurring at irregular intervals, and severe in type, caused the boy to roll about and cry out. Both recti tense. There was some interference with the functions of the bowels as they did not act as readily as in health. No urgent symptoms being present, it was decided to await further developments. If no improvement occurred in twenty-four hours an exploratory laparotomy was suggested, the suspicion tending in the direction of a mild inflammation of the appendix.

January 28th; temperature 100° F., pulse 104; P.M., temperature 99° F. Restless all night, pains in paroxysm continue. Two

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good fecal movements followed injections made by nurse. Dr. John F. Erdmann was now requested to see the patient in consultation and suggested an early exploratory operation.

January 29th; temperature 99° F., pulse 110, very restless and in pain all night. Bowels moved well (distinct fecal masses) after injection. The necessary arrangements having been made the child was sent to Gouverneur Hospital and operated upon by Dr. Erdmann at 3:30 P.M.

The subsequent details, kindly furnished by Dr. Erdmann, are as follows:

"When I first saw the little patient, Unger, with Dr. Joseph Huber, he had all the manifestations of an appendicitis without any shock. He had had some socalled results from enemata. I suggested to them that the boy be submitted to operation as soon as possible. He was admitted to the hospital the next morning for operation, and when under ether, upon abdominal and lumbar palpitation no tumor was found in the appendicular region, but upon subcostal palpation, a tumor about the size and absolutely the shape of a kidney was displaced, and could be brought down into the right iliac region. This tumor would slip back into the loin exactly as a floating kidney would. The abdomen was opened, with a view of exploration, through the right rectus. Digital examination detected a tumor involving the colon and cecum with a slight portion of the ileum. This tumor proved to be an intussusception, which was readily reduced; the appendix was removed, and the abdomen closed without drain. The patient made a perfect recovery, and was discharged to his home in ten days."

CASE II.—Nellie, nine years old, was seen in consultation with Dr. Hymanson. The history was briefly as follows: Four days before, without any apparent cause, the child was taken with severe pains in the abdomen, more intense in the umbilical region. The pains occurred in paroxysms, and at rather frequent intervals. She vomited several times daily; in the beginning the contents of the stomach were ejected, later on mucus and bile; no fecal odor noticed. As a rule constipation existed. It was reported that since her illness the bowels had acted several times in consequence of the cathartics which had been administered by the mother. No history of blood in the stools.

Physical examination: No heart or lung trouble. Liver and spleen normal. Abdomen retracted, both recti tense, pain and tenderness in the umbilical region; urination normal, rectal ex-

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amination negative. No tumor evident on deep palpation in the lumbar or right iliac region.

The similarity of this case to the one referred to above suggested intussusception and operation was advised.

The child was thereupon sent to the Beth Israel Hospital, to the surgical service of Dr. A. E. Isaacs, for exploratory laparotomy.

For the further history, I am indebted to my colleague, Dr. A. E. Isaacs, to whom I desire to extend my thanks for the courtesy of being allowed to see the case on several occasions.

The same evening the child was chloroformed and as nothing abnormal was discovered in the abdomen or per rectum, and as the symptoms were not urgent, it was decided to await further developments. The following day blood was observed in the stools, the same paroxysmal pains recurred, pain and tenderness still persisted about the umbilical region. The same evening a large quantity of fluid blood was passed. Temperature 99° F., pulse 100-110, respiration 20-24.

February 28th, 9 A.M., small movement (brownish fluid, with mucus and blood) very offensive. Still complained of pain. Temperature, pulse and respiration the same.

March 1st, operation by Dr. Isaacs. Medial incision. Intussusception in ileocecal region readily reduced. Appendix removed. The subsequent progress, except for some local pain and a small abscess in the deeper part of wound, was uneventful.

In the cases referred to above, rigidity of the recti muscles with umbilical pain and tenderness were present as constant symptoms. Vomiting occurred occasionally. The bowels could be moved by high enemata. Paroxysmal, colicky pains occurred in both cases. The suffering during the attacks was extreme, affording a striking contrast to the peaceful state in the interval. The temperature was slightly elevated and the pulse but little accelerated.

A tumor was detected in the first case when the child was under narcosis, just prior to the operation, that is, on the fifth day. In the second instance a thorough examination under chloroform failed to reveal any swelling. At no time was any blood or mucus present in the stool of the boy. In the case of the girl, characteristic bloody stools were first noticed after the child had been examined under chloroform about the sixth day of her illness. No collapse or evidence of shock in either patient.

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A detailed consideration of the salient points in the two histories may prove of value in assisting us in arriving at a positive diagnosis in future cases. Clinicians have for years recognized the import of *rigidity of the abdominal muscles with pain.* This point has been thoroughly discussed in an able article, from which, because of its importance, the following is abstracted: J. A. Blake (*New York Medical Journal,* January 3, 1903) emphasizes the value of rigidity as an early diagnostic point and believes that pain and rigidity go hand in hand as the cardinal subjective and objective signs of commencing as well as advancing abdominal trouble. "Rigidity is the reflex spasmodic contraction of the muscles of the abdominal wall exerted, not only to protect the irritated peritoneum from influences acting from without the body, but also to restrain the movements of the viscera and thus produce rest. It is an active, constantly-acting spasm of the muscle not necessarily accompanied with swelling or tenderness. Its degree and extent are valuable as evidencing a corresponding degree and extent of abdominal trouble. When slight it may be confounded with the voluntary contraction of muscles to protect a tender viscus and hence, the manner of eliciting rigidity is important. Gentle pressure with the flat of the hand, thus gaining the confidence and diverting the attention of the patient, will give much more valuable information than strong, deep pressure."

A tumor, when present, is a sign of great importance. It must not be forgotten, that we may not be able to demonstrate its presence until anesthesia is resorted to. Now and then, during the characteristic colic or while palpating the abdomen in a suspected case, an erectile mass may appear at the site of the lesion. If a tumor be present, it is apt to become more prominent under the condition referred to.

The pain is of two varieties. First, a constant pain with tenderness in the umbilical region; second, the recurring colicky attacks with evidence of extreme and acute suffering.

Text-books and clinical experience teach us that one or more of the diagnostic symptoms may be absent or not appear until late.

Paroxysmal colicky attacks in connection with *rigidity of the abdominal recti* and *localized pains* form a characteristic symptom complex. A consideration of these cardinal symptoms with

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the recent experience of the first case justified a positive diagnosis of intussusception in the second instance.

As a strange coincidence about this time, a girl of twelve years was admitted to the Beth Israel Hospital, presenting symptoms that were rather suspicious. A provisional diagnosis of intussusception had been made prior to her admission to the hospital. Her general condition was excellent. She had vomited some and had had severe abdominal pains, not paroxysmal in character, however. Pure blood had been passed per rectum. The abdomen was retracted and tender. The presence of a purpuric eruption upon the extremities revealed the true nature of the case and enabled us to exclude intussusception.

Appendicitis in children is frequently attended with pain in the umbilical region and rigidity of a rectus muscle. The pain, however, is of a different character, not necessarily paroxysmal. As a rule, there is but little difficulty in arriving at a proper diagnosis. The following instance, which occurred in the surgical service of my friend, Dr. H. M. Silver, at Gouverneur Hospital, is cited as an illustration.

N. L., *aet.* ten years, Russian. Schoolboy. Admitted January 18, 1903. History.—He has had no previous attacks. Digestion always good. About twenty hours before admission was seized with abdominal pains which in a few hours became worse on right side. Since beginning of attacks has eaten nothing, has vomited four times. Bowels moved twice in response to cathartics given at beginning of attack. Micturition normal. On examination right rectus muscle contracted, marked tenderness just below and to the right of umbilicus, very slight pain, and no marked rigidity of the muscles in the right inguinal region. No tumor felt. Abdomen somewhat distended, tympanitic. Temperature 103°F., pulse 138, respiration 30.

Operation.—Incision through right rectus muscle just below and to right of umbilicus; when peritoneum was opened a large quantity of seropurulent fluid escaped. Mass of congested omentum found just below incision. When unfolded, it was found to contain the tip of the appendix which was gangrenous; the appendix was directed upward and inward toward umbilicus and was four inches in length. Greater part of appendix and cecum healthy. Small contraction in apex of appendix. Wound closed, convalescence normal.

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LUMBAR PUNCTURE IN (*a*) OTITIC SEROUS MENINGITIS; (*b*) CEREBROSPINAL FORM OF TYPHOID FEVER; AND (*c*) CEREBROSPINAL MENINGITIS.

BY FRANCIS HUBER, M.D.,
New York.

Lumbar puncture of the subarachnoid space, proposed by Quincke in 1891, was demonstrated before this Society by Caillé in 1895. The following year, the subject was more fully discussed by him, particularly in reference to the local treatment of tubercular meningitis. Wentworth presented the results of "some experimental work on lumbar puncture," and at the same meeting C. G. Jennings reported a case. The method was urged principally for the purpose of diagnosis. In the summary of the experiments made at the Sears' Pathological Laboratory, Wentworth says: "The differential diagnosis between the various kinds of meningitis can be made by microscopic examination of the sediment, by culture taken from the fluid and by inoculation experiments." "Inoculation experiments afford the surest means of determining tubercular meningitis. It is of value to distinguish between the varieties of meningitis in order to determine if tubercular meningitis is recovered from."

In an excellent article (the first on the subject in this country), G. W. Jacoby (*New York Medical Journal,* December 28, 1895) writes: "Therapeutically, it is only of direct value as a palliative through the reduction of increased pressure: it may, perhaps, prove of more service indirectly as the first step to local treatment of the cord or brain."

A. Jacobi says, "In the treatment of tubercular meningitis lumbar puncture, mostly between the third and fourth lumbar vertebræ, has resulted in facilitating a diagnosis in many instances and in temporarily relieving some symptoms, those of congestion, edema, pressure, coma, but rarely aided in accomplishing a cure."

To quote further:—"In order to obtain any cerebrospinal liquor from the cranial cavity, lumbar puncture should withdraw from 10 to 15 c.cm. The total amount should not often exceed 20 or 30 at one time. None may be obtained when the needle

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does not reach the spinal canal, or is obstructed by fibrin,* or is caught between the nerve-fibres of the cauda equina, or when the connection between the ventricles and the cerebral and spinal subarachnoid spaces has been disturbed by exudative processes. The normal liquor, also that obtained in many pathological conditions, is clear, of 1007 specific gravity and contains from 0.2 to 0.5 per mille albumin. One per mille albumin in the fluid means inflammation. Sugar is sometimes found in connection with tumors. In simple *serous meningitis,* in chronic hydrocephalus, in that connected with pneumonia and other infectious diseases, also with uncomplicated tumors, the liquor is clear; in cerebrospinal meningitis it is turbid, somewhat purulent, and contains pneumococci and meningococci, in many cases (by no means all) of abscess and cerebrospinal meningitis pus and streptococci and staphylococci; in tubercular meningitis mostly bacilli; in apoplexies and hemorrhagic pachymeningitis blood. Kiliani and G. W. Jacoby found blood when the diagnosis of spinal hemorrhage had been made."

In his work on "Diseases of Children," Koplik tells us: "It is today one of the most useful adjuncts to the methods of diagnosis in acute and chronic forms of cerebral and spinal disease. Its future usefulness as a therapeutic measure is not clearly established, but will probably be in relieving symptoms due to pressure, and removing the excess of inflammatory exudate in the various forms of meningitis." On page 195 he advocates the procedure at first as a diagnostic and subsequently as a therapeutic measure. On page 196 he gives distinct and explicit indications for repeated puncture in cerebrospinal meningitis.

Most of the authors agree with Jacobi in the belief that it has not as yet given much satisfaction from the point of view of therapeutics. In meningitis it relieves the headache and the delirium and hyperesthesia, though the symptoms are liable to recur. In cerebrospinal meningitis and in lead encephalopathies favorable results have been reported by various authorities. Tubercular meningitis (the diagnosis having been confirmed by this method), we now know positively, may terminate favorably.

Several cases have come under personal observation, in which excellent results have followed the use of lumbar puncture. The most striking of these is the following that formed the basis of a

* "To the above may be added small particles of skin, fibrous tissue, bone or blood clot."

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paper read before the New York State Medical Society and reported in *American Medicine.*

CASE I.—Ida B., two and one half years old, born in New York, of Russian parentage, was admitted to the children's ward, Beth Israel Hospital, January 8, 1903. No history of tuberculosis in the family. History of insanity in grandmother, uncle died insane, aunt suffers from epilepsy. Father alcoholic.

Personal History.—Was born after a difficult labor, though instruments were not employed, cyanosed for several days, cry feeble. Breast-fed for nine months, began to walk at seventeen months. For more than two years has had a foul smelling discharge from the right ear, the original cause of which could not be made out at the time. No history of any eruptive fevers. A subsequent examination revealed large adenoids.

Present History.—Fourteen days before admission, the child became restless, began to cry a great deal and acted as though greatly frightened. Four days later began to have convulsions. The seizures were general and recurred every thirty minutes. The attacks were characterized by a loud cry, the child would fall to the ground suddenly. The movements were both clonic and tonic and lasted about three minutes. Of late, the attacks have increased in frequency, but are of shorter duration. Occasionally vomiting occurs after an attack, at other times the child would cry and fall asleep. The convulsive attacks occurred at night as well as during the day and then brought on by any external source of irritation.

Examination on Admission.—General condition was good. Face was suffused and expression dull; child apathetic and in a semicomatose state. Extremities were cold and blue. Surface generally mottled. Tongue coated, moist. Eyes convergent strabismus, moderate lateral nystagmus. Pupils dilated, right more so than left. Under light test contracted slowly and to moderate extent. Then slowly dilated again, and again contracted. Tache cerebrale was easily produced. There was an offensive purulent discharge from the right ear, no tenderness over the mastoid region or side of skull; no edema of this area. Pulse rapid, no irregularity or intermission. Reflexes in general exaggerated. Moving the patient or attempts at feeding were followed by convulsive seizures. Lumbar puncture proposed for diagnostic purposes, could not be carried out as the violent character of the attacks

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brought on in attempting to put the child in position, interfered with the necessary manipulations.

The neuropathic family history of insanity, epilepsy, etc., in the parents and near relatives, suggested a condition of status epilepticus. The latter, however, could be excluded from the history of the case, and the fact that the convulsions dated back only two weeks. Careful inquiry failed to elicit a history of a fall, blow or traumatism of any kind. By exclusion, therefore, we were compelled to look upon the chronic suppuration in the ear as a probable etiological factor, though no focal symptoms were present. The statistics of Pitt estimate that 5 per cent. of all cases of meningitis are of otitic origin. Dr. W. Freudenthal, to whom I herewith extend my thanks for his wise and conservative counsel, was requested to examine the patient.

Report of Dr. Wolf Freudenthal. "On January 10, 1903, I saw the above mentioned child at the request of Dr. Francis Huber. She had had convulsions, which were increasing in number and severity, the pulse was getting irregular and the whole aspect of the case appeared worse than at the beginning. There being a suspicion of mastoiditis I was called in. After removing from the right ear the offensive discharge that had lasted for the past two years, an exceptionally large perforation of the drum membrane could be seen, out of which pus oozed. There was no swelling over the mastoid, no tenderness or pain over any part, the temperature was 100° F., pulse 130 and the child cried incessantly. Besides it had had seven attacks during the past night and eight during the day. As the mastoid symptoms were not very well marked, it was thought there could be no risk in waiting twenty-four hours longer. At the end of this period the attacks had increased in frequency and the general prostration was more marked. The general belief of all concerned in the case now was, that the only salvation for the child could come from the opening of the mastoid. At 2:30 P.M. on January 11th the operation was performed in the usual manner. After chiseling away all the diseased bone, the dura mater was seen to bulge outward and it was apparent that there was pressure from within. I hesitated, however, to withdraw any fluid, because an infection was possible and surely not desirable. The wound was packed and at 4 P.M. the house physician, Dr. Nisonoff, under the supervision of Dr. Huber, withdrew about 30.0 grams of spinal fluid by means of lumbar puncture. Within the next two hours the child had

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three convulsive attacks. Six hours later twitching of the extremities set in beginning at 12 midnight and ending at 1 A.M. She became quiet after repeated hypodermic medication. Another such severe attack commenced at 4:45 in the morning and lasted for forty-five minutes. At noon the next day (12th) 16.0 grams of spinal fluid were withdrawn, after which the symptoms slowly but gradually improved and the child was discharged on February 2, 1903, *i.e.*, twenty-two days after the operation was performed." When seen subsequently by the writer, about two months later, she was bright and active, running about and playing, presenting no evidence of her severe illness.

In the treatment of the case there is no doubt in my mind that in spite of the nonexistence of any local evidence of mastoid trouble, the operation was justified. In addition to getting rid of the local diseased focus we were enabled to eliminate a possible pachymeningitis and at the same time relieve the pressure to a limited extent. In resorting to lumbar puncture an accidental infection immediate or remote of the cerebral meninges was avoided. The favorable therapeutic effects following the withdrawal of the cerebrospinal fluid were manifested in a very few days. On the 14th the child was still somewhat restless. On the 16th she was bright, took an interest in her surroundings and could with difficulty be kept in bed.

The following case is presented with some hesitation and many apologies, not because of any doubt as to the therapeutic value of the puncture, but rather in view of the facts that a more careful bacteriological examination of the cerebrospinal fluid had not been made. The fluid was sent to an expert pathologist and bacteriologist, who reported a negative finding as regards tubercle bacilli. Unfortunately no cultures were made, nor was there any examination for typhoid bacilli instituted, though such a request was made when the specimen was sent to the laboratory.

CASE II.—Annie H., nine years of age, school girl, born in Russia, eight years in this country, was admitted to the Beth Israel Hospital, October 4, 1902. The family and personal history, not having any bearing upon her present trouble, is omitted.

Present History.—About four days before admission, the child began to complain of headache; constipation and elevation of temperature were noticed. The fever which was quite high, has persisted. A careful inquiry failed to elicit a history of chills or convulsions.

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Examination on Admission.—The child was in a semicomatose state, very restless, with slight twitchings of the muscles of the face and extremities. The head was retracted. Pupils were dilated and did not react; nystagmus was present, but no strabismus. She had vomited some and was quite delirious. Extremities were cold. Kernig's sign present. Tache cerebrale readily brought out. Heart and lungs negative. Abdomen distended and somewhat tender. Roseola fairly numerous. Temperature 105°F The pulse was weak, no irregularity in force or rhythm. During the following three days marked rigidity of the extremities and neck were noticed. The pupils were dilated and reacted in a sluggish way to light. Hydrocephalic cry. Marked muttering delirium.

October 6th.—Pulse very weak, child somewhat cyanosed.

October 7th.—As the delirium had increased and the headache had become more violent with frequent outcries as though suffering intense pain, it was decided to resort to lumbar puncture. About two drams of clear cerebrospinal fluid, were allowed to escape, the fluid flowing in a steady stream with considerable force. After this the pressure symptoms were somewhat lessened. Widal's reaction positive (1-20). Incontinence of urine was present. Swallows with difficulty.

October 8th.—Child unconscious; pulse very weak; patient lies in bed with knees drawn up; extremities rigid.

October 9th.—Semicomatose condition, though at intervals is apparently rational. Pupils respond slowly and imperfectly—lumbar puncture negative or rather unsuccessful.

October 10th.—Child shrieked constantly toward morning, and was delirious; urinates involuntarily; was rational most of the time during the day. Subdural lumbar puncture made today and two drams of fluid escaped with evidence of considerable pressure.

October 11th.—Widal's reaction positive (1-20). Rigidity of extremities marked, more so in lower.

October 12th.—Child rational greater part of the night. Pupils react but little; shrieks during sleep.

October 13th and 14th.—More rational and quiet. Examination of fundus of eye negative.

October 16th-22nd.—Pupils still dilated, reacting but sluggishly to light.

October 24th.—Widal's reaction positive (1-20).

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October 27th.—Irregular reaction of pupils; left less than right.

October 31st.—Dr. J. E. Weeks' examination of the eyes revealed some slight anemia of the fundus, otherwise normal.

November 3d.—General condition greatly improved.

November 4th.—Pupils responded well to light, but are still dilated.

Improvement continued and on 21st child was allowed to sit up in the ward. No further untoward symptoms having in the meanwhile developed the child was discharged in excellent mental and physical condition about November 30th.

Temperature on October 4th, on admission 105° F. October 4th-9th, ranged between 103°-106° F., sponge or pack reducing it but slightly. October 9th-14th, ranged between 100°-105° F., there being an evening rise with big drops after sponging or packing. October 15th-25th, varied between 100° and 103° F., with regular evening exacerbations. October 26th to November 6th, from 99° to 102° F., 5 P.M. November 6th-9th, 100° F. or below. November 10th-21st, regularly less than 100° F. November 21st-29th, from 98° to 100° F.

Urine.—Negative throughout.

Pulse.—Irregular in force at times, without irregularity in rhythm.

Respiration.—For the first few days from 30-36. Later on about 24.

Of course in the case reported above, it may be questioned whether there was any relation of cause and effect between the removal of the cerebrospinal liquor and the recovery. In this connection, we must not fail to consider that cases presenting marked symptoms of meningeal irritation are not all necessarily cases of meningitis. We are fully aware that cases occur in different acute infectious diseases (typhoid, influenza, pneumonia, etc.) in which anorexia, vomiting, constipation, insomnia, delirium, severe pains in the head and neck with cervical stiffness and unequal pupils are observed, which recover completely in a very short period of time.

Hutinel in discussing nonsuppurating meningitis (*Revue mens. des Mal de l'Enfance,* Vol. XX., No. 4) writes as follows:

"The meninges, like any other organ, may react to an irritant in varying degrees from a passing congestion to suppuration. The dividing lines established between meningitis, serous meningitis

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with bacteria and that without bacteria are purely theoretical.* Serous meningitis almost always occurs in the course of grave infections and has no pathognomonic symptoms. The onset may be sudden, or insidious and unrecognized. Vomiting is more constant than constipation.

Osler writes:—"In rare cases of typhoid the disease sets in with most intense cerebrospinal symptoms, simulating meningitis, severe headache, photophobia, retraction of the head, twitching of the muscles and even convulsions, occasionally drowsiness, stupor and signs of basilar meningitis may exist for ten days or more before the characteristic symptoms develop. The outset may be with mania."

Meningitis, though extremely rare, may occur. With the evidence obtained by lumbar puncture and the presence or absence of Kernig's sign, the diagnosis is now more easily made.

P. Moizard and H. Grenet (*Arch. de Méd. des Enfance,* June, 1903) report an instance of the cerebrospinal form of typhoid fever in a boy five years old. Such cases are relatively rare according to these observers. In the article the literature is fully quoted. Lumbar puncture failed to reveal any cellular elements.

In the case just reported, which recovered and in another which terminated fatally several weeks later, there was certainly an increase in intracranial pressure as shown by the forcible escape of the cerebrospinal liquor when lumbar puncture was made. In this connection the following observations are of interest. Nobecourt (*Revue des Mal. de l'Enfance,* Vol. XXI., No. 4) discusses the pathogenesis of the meningeal symptoms frequently observed in the course of bronchopneumonia—nontubercular in origin. He believes that the condition is better understood since lumbar puncture was regularly practiced.

The author finds that when meningeal symptoms are present, there is usually an *increase* in the amount of cerebrospinal fluid. It is usually limpid; exceptionally it is turbid. The fluid is generally sterile, now and then pneumococci are found.

J. H. Musser in a paper on "Therapeutic Value of Spinal Puncture for Cerebral Symptoms in Typhoid Fever," read before the Congress of American Physicians and Surgeons recently, states that he has noticed symptoms, which the French termed

* It is a mistake not to admit that there may be transitory and curable meningeal irritations.

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meningismus, and which disappeared after a lumbar puncture was performed.

CASE III.—I. C., two-and-a-half years old, born in United States, admitted May 24, 1903, to the children's service, Beth Israel Hospital.

Family History.—Parents living and well. No diathesis, tubercular or rheumatic.

Personal History.—Labor normal. Bottle-fed (milk and prepared barley) for eight months, then mixed diet. Began to walk at fifteen months. Dentition normal.

Previous History.—No discharge from ear. Had diphtheria twelve months ago. One week later nephritis; also complicated by pneumonia. Whooping-cough six months ago and measles three months later.

Present History.—Began five days ago with fever, projectile vomiting, restlessness and headache. The vomiting continued for the first two days. The headache seems to have increased in severity as shown by facial aspect of pain. Three days ago the mother noticed retraction and stiffness of head; anorexia. Bowels constipated. Urine diminished.

Temperature on admission 103°F., which declined gradually to normal on third day. Since then has been between 99° and 100° F.; pulse varied between 130-120; respiration between 32-20.

Urine Examination.—May 30th and June 9th, light color, clear; acidity slight; specific gravity 1018; no albumin or sugar; microscopically—negative.

Physical Examination.—Child well nourished. Apathetic and semistuporous. Head retracted and rigid; tongue heavily coated; breath foul, due to ulcerative stomatitis; there are few ulcers on hard palate. The lips were fissured, on the inner surface numerous minute ulcers are found. Herpes labialis present. Nose, ears and mastoid normal. No nasal discharge. The pupils reacted to light; left was more dilated. Expression dull with a vacant stare; photophobia; pulse varied in force, frequency and regularity; heart normal; lungs negative; abdomen distended, showing stains of an old eruption. There were numerous superficial ulcers over lower end of sacrum and trochanters, probably the result of scratching. Reflexes are absent. Kernig's sign was obtained as well as cerebral tache.

May 25th.—Lumbar puncture; nothing could be obtained.

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May 26th.—Lumbar puncture was again tried. This time eleven drams of a somewhat cloudy fluid removed. Microscopically a very large number of pus cells and few mononuclear cells were found. No tubercle bacilli or other microorganisms.

May 29th.—General condition. The child is much brighter, takes interest in things about him. The pupils are equal and react slowly. Pulse is regular.

May 30th.—Leukocytosis only 9,000.

June 1st.—Improving. Stomatitis also improving. Pupils equal and react sluggishly to light.

June 4th.—Child tries to sit up. Brighter.

June 6th.—Although the general condition is better the expression is still somewhat dull and stupid. No strabismus. Lymph nodes beneath angle of jaw enlarged. The ulcerative stomatitis is improving. Upper two incisors necrotic. Tongue is slightly coated and moist; heart and lungs negative; liver and spleen not palpable; abdomen distended, soft. Tache cerebral present; Kernig's sign absent. Superficial and deep reflexes present. Extremities cold; no edema or cyanosis.

June 10th.—Child sits up. Interested in things about him. A few days later discharged in good condition.

In the above case a single large tapping sufficed to relieve the tension, thereby favoring absorption. In another case at Roosevelt Hospital, the child's condition was very bad. Convulsions recurred at frequent intervals. Repeated tappings of small quantities were resorted to. The child recovered ultimately, but the mental faculties were left greatly impaired.

In the *Medical News,* March 23, 1901, Koplik reports a series of cases of cerebrospinal meningitis and advises repeated lumbar puncture as a curative method of value.

Other cases are reported in literature in which not infrequently a single tapping only was required. In the greater number, however, repeated punctures were necessary.

Though the cases reported by the writer are but few, they came under observation in a relatively short time. The results in each case were gratifying and tended to strengthen the favorable impression as to the therapeutic value of the method.

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A CASE OF TALIPES EQUINOVARUS OPERATED ON BY PROFESSOR LORENZ.

BY SAMUEL S. ADAMS, A.M., M.D.,
Washington, D. C.

Through the courtesy of Dr. J. Ford Thompson, the surgeon of the Children's Hospital, Washington, D. C., I am permitted to present the boy, whose record is here given. A short time before Professor Lorenz operated on this boy I exhibited him before the Medical Society of the District of Columbia, as the worst case of talipes equinovarus ever treated in our hospital. At that meeting Dr. Thompson explained the several operations he intended to perform, among which was the removal of a wedge of bone from the outer side of each foot. He explained the unpromising features of the case and was doubtful as to a *good* result. The casts and skiagraph (see Figure I.) show the condition of the feet before the Lorenz operation, while the patient himself presents the best evidence of success as he stands and walks on over-corrected feet. In a short time shoes will be fitted and he will leave the hospital.

C. W., ten years of age, male, white, admitted to Children's Hospital, November 15, 1902.

Family history negative.

Previous History.—Labor normal; breast-fed. Dentition normal. Has never had any of the usual diseases of childhood. He has always been a bright, healthy, active child.

History of present illness dates from birth. Exaggerated case of talipes equinovarus.

Present Condition.—General appearance:—Height, very good for age; posture, negative; facial expression intelligent. Form and symmetry of body:—Upper extremities are short, the bones, however, being very well developed. The hands are thick and the fingers are thick and short, being club-shaped. The lower extremities are thick and short, the toes being clubbed. The feet are noticeable chiefly for the very pronounced case of talipes equinovarus. Each foot is turned toward the median line to such a degree that in walking the patient is compelled to raise it quite high in order to prevent it from striking its fellow. The heel is

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raised so that by a combination of the two contractures he walks on the anterior external portion of each foot, which has greatly thickened the skin on the outer side of each little toe.

Other systems all normal.

November 25th.—Patient prepared for operation. Chloroform administered. The tendo Achillis of each foot was severed and the attachments of the muscles on the external surface of the foot

FIG. 1.—SKIAGRAPH OF CASE OF TALIPES EQUINOVARUS BEFORE OPERATION.

were also cut. (The notes are not very clear as to the exact character of this operation.)

December 5th.—Patient prepared for the operation which was performed by Professor Lorenz at the Providence Hospital. The attachments of the ligaments were destroyed by manipulation. Each foot was restored to an over-corrected position and covered with thick coats of cotton and plaster of paris.

December 5th.—Patient returned to the hospital after the operation suffering much pain. Morphin sulphate gr. ⅛ was given hypodermatically. He slept very well during the night.

December 6th.—Pulse is weak toward morning and strychnin

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sulphate gr. $^1/_{100}$ and whiskey ʒiv were given every three hours. Patient suffered much pain in the morning and evening. In each instance morphin was given hypodermatically.

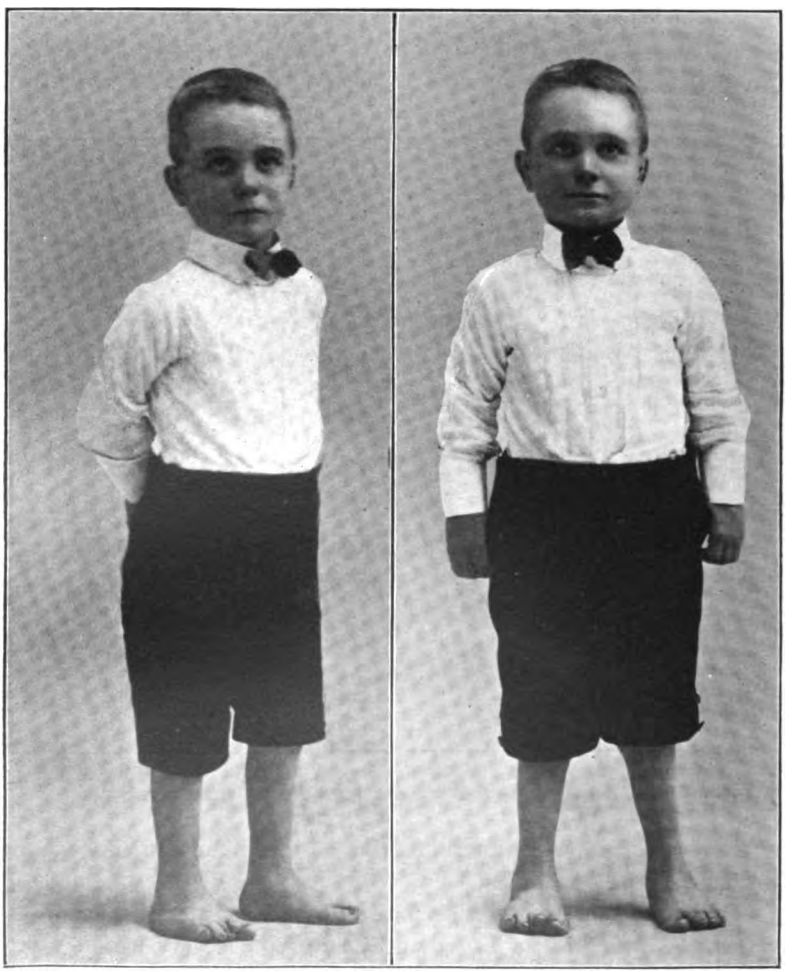

FIG. II.—PHOTOGRAPHS OF BOY FOURTEEN MONTHS AFTER OPERATION.

December 7th.—Patient slept only after hypodermics of morphin, which were given at 1:30 A.M., 3 P.M. and 10:45 P.M. At times his suffering was intense.

December 8th.—Pulse weak and irregular. Stimulants im-

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proved this condition somewhat. Patient suffered greatly. Morphin given at 1 P.M. and 8:50 P.M. Patient enjoys his milk, taking full amount.

December 9th.—Morphin administered at 7:25 A.M. and 1:30 P.M.

December 10th.—Pulse weak and irregular. Responded to stimulants. Morphin at 1:30 A.M., 1 P.M. and 8 P.M.

December 11th.—Patient still suffers greatly. Morphin (gr. ⅛) at 11 A.M and 11 P.M.

December 12th.—Patient rested better. Morphin at 6 P.M.

December 15th.—Suffered very little. Not necessary to give morphin. Regular diet. Pulse much stronger. Appetite splendid.

December 19th.—Allowed to get up in rolling-chair. Does not complain of pain.

January 1st.—Improving. Suffers no pain.

February 1st.—Walks a little with assistance.

February 6th.—Is able to walk without assistance.

February 25th.—Walks much better.

March 1st.—Walks well.

May 1st.—Casts removed. Feet found to be in normal condition.

For a week afterward he complained of soreness of the soles of his feet. He had some difficulty in walking, but in ten days he was walking without assistance and has steadily improved.

I present herewith photographs of this boy (see Figure II.), which were taken fourteen months after the operation. He walks well, but the foot is still very flat.

DISCUSSION.

DR. ADAMS.—The treatment required was about twenty minutes with both feet. He has worn a plaster cast for three months. No cutting was done. The point where the tenotomy had been performed ruptured and bled a little from the tendo Achillis. The operation was simply manual. You will notice that there is some over-correction. Dr. Lorenz preferred to put on the cast and have over-correction. The subsequent treatment will be that he will be put in a cast and remain that way for several months longer, and I think at the end of a year a shoe will be fitted to him, a scientifically constructed shoe.

DR. ROTCH.—I have at present the medical care of one of Dr. Lorenz's cases, a congenital hip, which has been in plaster for

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seventeen or eighteen weeks. When the patient first came under my care she had scarlet fever and we had to take the cast off in order to see the condition of the skin under it; she will have to be kept in the cast for perhaps two or three months more. It is a little girl about twelve years old. In this case of scarlet fever the skin under the cast looked very well.

DR. ADAMS.—When the cast which was on this child was taken off the callous which was on there peeled off and there is practically new skin. Oil was first put upon his feet, then scrubbed off with soda to get the skin in good condition before putting on another cast, but it is the intention of Dr. Thompson to put the cast back as directed by Dr. Lorenz. He will stay in that cast for the rest of the year and then have shoes put on. The most remarkable thing was that this boy could walk about with the casts on as if nothing were wrong with him at all, and before the operation he was always falling about.

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